ARCHIVES OF OPHTHALMOLOGY.

THE NATURE OF SO-CALLED KOEPPE NODULES.[1]

By Dr. GEORGE S. DERBY, Boston, Mass.

(With two illustrations on Text-Plate XXXIII.)

E. F. K., 43 years of age, was seen on February 2, 1927, complaining of failing vision in the right eye for two years past. There was no history of trauma and he stated that his general health was excellent. In 1910 he had been run down and sent away for four months, but returned in good condition. The examination of the eyes was as follows: Vision right: $\frac{5}{200}$ not improved. Vision left: with glass $\frac{11}{18}-$. Right eye was not injected, but showed many precipitates on the posterior surface of the cornea. The iris was somewhat discolored. The lens was cataractous and only a very dull red reflex could be obtained. With the corneal microscope and slit lamp there were some precipitates on the posterior surface of the cornea and many cells in the anterior chamber. The iris was studded with many translucent (Koeppe) nodules which were on the pupillary margin, in the tissue running back to the lesser circle, and beyond. The iris margin and tissue showed some atrophy and there were a few visible vessels.

The left eye was normal anteriorly, the lens was clear. There were floating vitreous opacities, and above the disc and temporally there was an area of choroidal atrophy, with a small veil-like membrane extending into the vitreous.

The patient was sent to Dr. J. B. Hawes, 2d, who found by physical examination and X-ray much scarring at the top of each lung. While under observation by Dr. Hawes the process became slightly active and produced a slight

[1] Read before the American Ophthalmological Society, Washington, D. C., May, 1928.

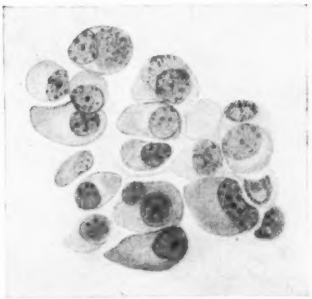
increase in temperature and rapid pulse. The sputum was negative, but Dr. Hawes made a positive diagnosis of pulmonary tuberculosis. As some medical man had told the patient that his eye condition was due to syphilis he was sent to Dr. Austin Cheever, who ruled it out as there were no signs of the disease, the blood was negative to numerous tests, and the spinal fluid was negative to one.

On January 3, 1928, as the right lens had become totally opaque and the light projection was good I did a combined extraction, obtaining a vision in the eye with correcting glass of from $\frac{1}{10}$ to $\frac{1}{10}-$. When seen last on March 20th the right eye still showed precipitates on the back of the cornea, cells in the anterior chamber, nodules in the iris and on the pupillary margin, a few vitreous opacities and a normal fundus.

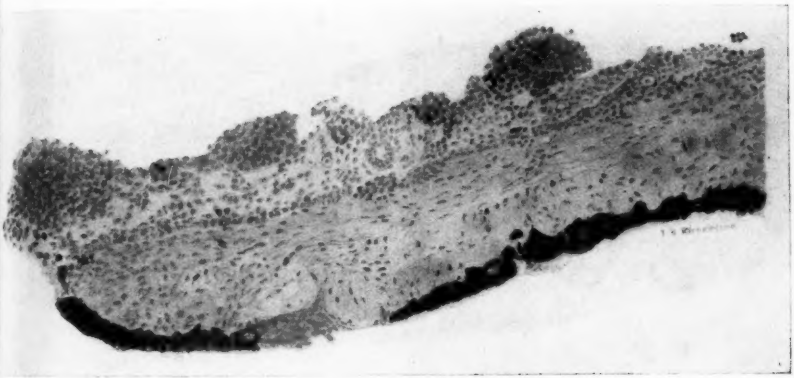
The excised piece of iris, which had shown numerous nodules with the corneal microscope, was examined by Dr. Verhoeff, who reported as follows: "The section consisting of a small piece of iris was . . . sectioned serially. . . . On microscopic examination the iris is found to be free from tubercles and lymphocytic nodules. Its stroma is considerably infiltrated with plasma cells, but it is not otherwise notably altered. In relation with the anterior surface there are many nodular collections of these cells. Some of the nodules lie on the surface, some are situated partly within the stroma, and some are at the pupillary margin. Many of the cells within the nodules are somewhat atypical, but can still be recognized as plasma cells by the eccentric situations of the nuclei and the clearer spaces in front of the nuclei. The largest nodule is .13*mm* in diameter."

In 1917 Koeppe described these nodules in connection with tubercular iritis, believing them to be a valuable indication of the disease. Since that time they have been found also in sympathetic disease, in leprosy and in so-called "tropical syphilis." Whether they occur in other conditions is not as yet settled. They are transient in character, appearing and disappearing within a short time. Probably they may at times lead to the formation of synechiæ. In my experience these nodules are very common in low-grade uveitis, and it is my belief that in most of these cases their presence should give rise to the suspicion of a tuberculous etiology. They have not been found in the ordinary form of syphilitic iritis which we see, or in iritis supposedly due to focal infection.

ILLUSTRATING DR. GEORGE S. DERBY'S ARTICLE, "THE NATURE OF SO-CALLED KOEPPE NODULES."



Individual plasma cells of which the nodules were composed.



Section of iris showing collection of plasma cells on the surface and in the stroma.

In most cases of serous iritis a search for a definite etiology is usually fruitless, but in the case reported here the patient had the undoubted signs of a pulmonary tuberculosis of which the lesions were largely healed.

The condition of the left eye is of interest in connection with Verhoeff's paper, also to be presented at this meeting. In E. K.'s left eye there was a single healed area of chorioretinitis and vitreous opacities. At some previous time he had had an acute localized choroiditis with exudate in the vitreous and quite possibly a slight anterior involvement, the traces of which had completely disappeared. It is reasonable to believe that the choroiditis was due to the same etiology as the disease in the right eye, namely, in all probability, tuberculosis, and this is consistent with Verhoeff's views as to the causation of these choroidal patches, with which views I entirely agree.

The histological picture of these so-called Koeppe nodules is of extreme interest, and especially in this case where they were present in such great numbers. The fact that they were entirely composed of plasma cells is of no great significance, but quite consistent with an early stage of tuberculosis caused perhaps by action of a toxin. I have been given to understand that a microscopic description of these nodules has been published by Gilbert, but I have been unable to find his article in the literature.

It seems desirable that, whenever possible, pieces of irides which have shown these nodules should be excised for examination, and such opportunities should not be very infrequent. The advent of the slit lamp and microscope has shown the frequency with which cataract, especially the monocular form, is due to a low-grade uveitis. This cataract is operable and good results, at least for a considerable period of time, are usually obtained.

VISUAL ACUITY AND ILLUMINATION.[1]

By SELIG HECHT.

(From the Laboratory of Biophysics, Columbia University, New York City.)

(With six illustrations in the text.)

I

WE are all familiar with the fact that visual acuity in the normal eye varies with the illumination. This has been known probably from earliest times. It was first investigated in 1754 by the astronomer Tobias Mayer, who believed to have found that visual acuity varies as the sixth root of the illumination intensity. Since Mayer's day many investigators such as Klein, Posch, and Cohn tried to find the exact relation between visual acuity and illumination. The best of this early work is by Uhthoff who studied not only white but colored lights at various illuminations as well.

A few years later (1897) Koenig redetermined the influence of illumination on visual acuity in such detail that his data have become classic. They are reproduced in Fig. 1. All the experiments since then have only served to confirm Koenig's data. Since it is proposed to use these data as a basis for theoretical study of the relationship between illumination and visual acuity, it may be well to give the recent data of Roelofs

[1] Abstract of lecture given to the Section of Ophthalmology of the New York Academy of Medicine on April 16, 1928. The complete data and a detailed treatment of the subject here considered will be found in a paper in the *Journal of General Physiology*, 1928, xi, 255, which contains as well all the references to the literature.

and Zeeman also. These are presented in Fig. 2 from which it is apparent that they are an excellent corroboration of Koenig's work.

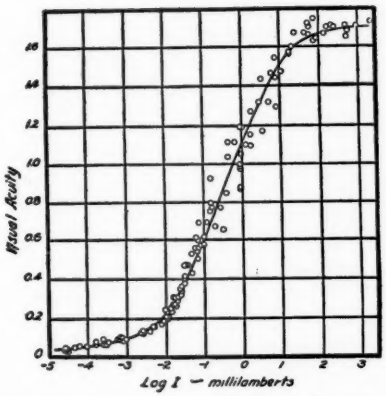


FIG. 1. Relation between visual acuity and illumination. Data of Koenig (1897).

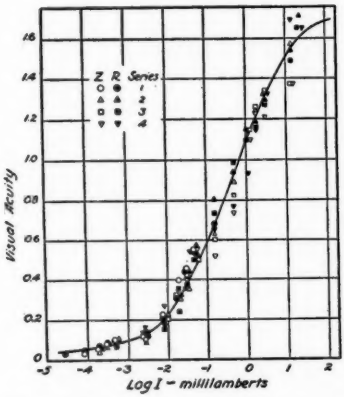


FIG. 2. Relation between visual acuity and illumination. Data of Roelofs and Zeeman (1919).

In spite of the familiarity of this information and the accuracy of the data there exists no explanation for this

dependence of visual acuity on illumination. It is with an effort to explain this relationship that the present paper is concerned.

II

The fineness of detail which a surface, such as for example the photographic plate can register, depends on the number of receiving elements present in a unit area of the surface. In other words its resolving power varies inversely with the average distance between the sensitive elements. The retina is a surface of this kind since it is composed of discrete rods and cones which function as units or groups of units. The way in which visual acuity varies with illumination indicates the way in which the resolving power of the retina varies. A low visual acuity means that the average distance between the retinal elements is large, whereas a high visual acuity means that the distance is small. To account in such terms for the large variation of visual acuity with illumination, one must suppose that the number of sensory elements per unit area of retina can and does vary nearly a hundred fold. But this is nonsense from the anatomical point of view, because the number of rods and cones is fixed structurally. To get around this it is therefore necessary to assume that the number of these elements is variable functionally.

Let us suppose that the individual rods and the individual cones do not all possess the same sensitivity to light. Some will be stimulated by low illumination, others only by higher illumination. Then at low illumination the resolving power of the retina, and therefore visual acuity, would be low because there are fewer sensory elements working per unit area. Moreover as the illumination increases and more and more retinal elements reach their threshold of stimulation, visual acuity would continue to increase until finally all the elements are functional and no further increase in visual acuity is possible. This of course is precisely the way in which the phenomenon takes place.

III

It is possible to make this hypothesis strictly quantitative so that it may be compared directly with the accurate numer-

ical data of Koenig and of Roelofs and Zeeman as given in Figs. 1 and 2. Before this can be done, however, it is necessary to consider one variable which was not controlled in these measurements, namely, the size of the pupil. This varies with the illumination, and if ignored, gives an erroneous idea of the actual illuminations on the retina which result from the measured outside illuminations.

A correction for this variable is possible in terms of the measurements of Reeves. Fig. 3 is computed from Reeves'

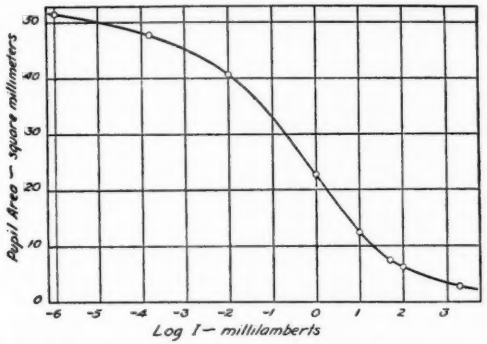


FIG. 3. Relation between pupil area and illumination, as calculated from the data of Reeves (1918).

data and gives the relation between pupil area and illumination. The outside illuminations multiplied by the pupil area in square millimeters gives the resulting illuminations in terms of photons, or units of retinal illumination introduced by Troland. It is these corrected data which we shall now proceed to analyze quantitatively.

IV

There are several million rods and cones in the retina. These may be considered as composing two populations: one, the rod population, sensitive to low illuminations and occuring outside of the fovea; and the other, the cone population sensitive to higher illuminations, receptors for color, and situated all over the retina but very densely in the fovea.

If these two populations are like all other biological populations with which we are familiar from the work of statisticians, then their variation in sensitivity to light should show the usual bell-shaped distribution curve.

Fig. 4 shows the kind of frequency distribution curves for the thresholds of the individual rods and cones which have to be assumed in order to describe quantitatively the corrected data of Figs. 1 and 2. It is apparent that these curves are similar to the usual frequency curves of the biometricians.

The curves in Fig. 4 give the frequency distribution of the *thresholds*. What we wish to know, however, is the *total*

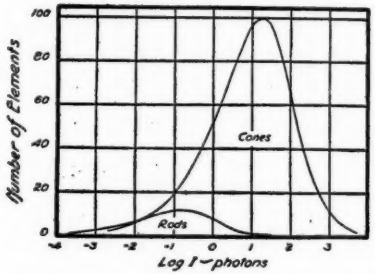


FIG. 4. Distribution of thresholds of rods and cones. The two curves are identical in form but different in position and in size of ordinates.

number of rods or cones which are functional in a unit area in any given illumination. This can be done by integrating the curves in Fig. 4. In non-mathematical language this means finding at any intensity the area under the curve to the left of that intensity. When this is done, the two curves of Fig. 5 are secured. These give the total number of rods or cones which are functioning per unit area at any given intensity. It is apparent that these two curves resemble the visual acuity data in Fig. 1 and Fig. 2.

Beginning with the lowest illuminations vision is a function of the rods. The number of rods which are active is very small; this amounts to having a resolving surface with the receiving elements sparsely distributed. The retinal distance between two just discriminable contours must be large, and visual acuity is very low. As the illumination increases, more

and more rods reach their threshold and become functional, with a resulting increase in visual acuity. Presently an illumination is reached when the first cones begin to function. Visual acuity will still be mediated by the rods because there are still more active rods present than cones. But as is apparent from Fig. 5 the rate at which the cones come into play with increased illumination is nearly ten times as great

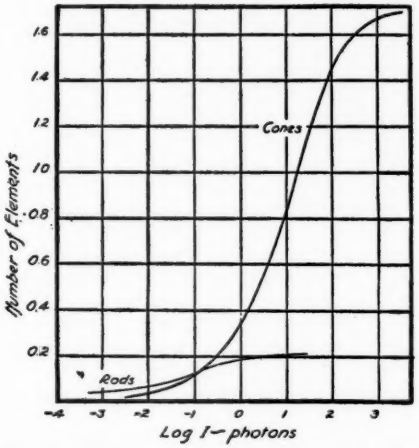


FIG. 5. Relative number of rods and cones which are functional at any illumination. The curves may be described by the common Gram series of the statisticians.

as the rate of the rods. Therefore at a certain point the number of cones functioning in the fovea will be equal to the number of rods in the periphery. As the intensity increases beyond this, the number of active foveal cones per unit area will be greater than the number of active peripheral rods in a unit area, and visual acuity will now be determined by the cones. This augmentation of the number of functional cones and the resulting rise in visual acuity will continue until all the cones are active and no further change in visual acuity is possible.

The curves in Fig. 5 are corrected for pupil area. To show how accurately they describe the originial data of Koenig and

of Roelofs and Zeeman the lines in Fig. 1 and Fig. 2 are drawn. These lines are derived from the curves in Fig. 5, and are transferred back to the original uncorrected data by means of the pupil area curves given in Fig. 3. It seems clear that the theory here proposed, as expressed in the curves drawn through the experimental points in Figs. 1 and 2, is an adequate quantitative description of the relation between visual acuity and illumination.

V

There are several implications from these data, and I propose to consider a few of them. The data heretofore

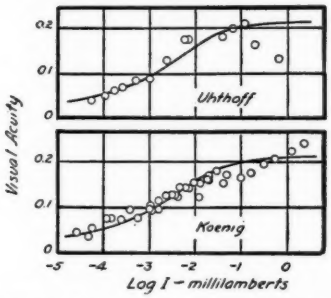


FIG. 6. Visual acuity of two completely color-blind individuals. Compare this figure with Figs. 1 and 2, but note that the ordinates here are twice as large as those in Figs. 1 and 2.

given are for the normal eye, and the proposed explanation obviously rests on von Kries' theory of the functional separatness of rods and cones. Koenig recognized this when he attributed the lower limb of the data to the rods and the remainder to the cones. He also recognized the implications involved namely, that in a completely color-blind eye the cone portion of the curve should disappear and leave only the lower rod limb plus any extensions of it. Fig. 6 gives the data for two such individuals, one measured by Uhthoff and the other by Koenig. These bear out the supposition that the two limbs of the normal curve follow von Kries' duplicity theory.

These data are equally significant for our explanation.

Fig. 5 shows that for the normal eye the cones overtake the rods at the middle of the rod range. It therefore follows that in a completely color-blind eye the visual acuity data should extend to a distance beyond the rod-cone intersection point equal to that which has preceded it. Moreover, the entire visual acuity data of such a case should be described by only the rod curve of Fig. 5. The lines drawn in Fig. 6 are this rod curve taken from the theoretical curve of Fig. 5 and corrected for pupil area as already explained.

It is apparent that both suppositions are correct. The data of the completely color-blind extend over twice the range of the rod vision of the normal. There is no rod-cone inflection point, and the entire data are describable in terms of the rod distribution curve alone.

It must be apparent that in the complementary case of a night blind person the rod portion of the curve should be lacking and the entire visual acuity curve should be describable by the cone curve alone. Such a case has not been studied in this way, but it is confidently expected that this will be the result.

VI

In Fig. 5 the number of elements functional per unit retinal area was arbitrarily chosen to read directly in visual acuity units. The real number of elements will depend on the size of the unit area which is selected. Is there really such a thing as a unit retinal area? Is there a minimal retinal area which can carry out all the functions of the retina as a whole? Such a unit area should be able to see the entire range of colors, to cover the whole field of intensity discrimination, and to perform the total visual acuity function. If the retina is indeed composed functionally of such minimal areas, then the size of this area, as found from the data of the three visual functions enumerated, should be comparable.

Working with brightness discrimination, Koenig found that the whole range of intensities visible to the eye is made in only 572 discrete steps in intensity recognition. About 30 of these steps are mediated by the rods; the rest, 542, by the cones. This is fact. In terms of our notion of the sensibility distribution of rods and cones, the recognition of an intensity

difference corresponds to a change from n to n plus 1 or to n minus 1, elements functioning in a unit area. Therefore the minimal retinal area must contain 542 cones, or some multiple of it.

In the same way a minimal retinal area should be able to mediate the whole range of visual acuity. The lowest visual acuity represents the widest retinal distance by which the images of two contours must be to be recognized at the lowest illumination visible. Within this largest retinal distance all other visual acuities are contained. It must therefore represent the side of a square whose area is the minimal retinal area. The lowest visual acuity in Fig. 1 is 0.03 unit. This is a visual angle of slightly over 44 minutes and is a distance of 0.2*mm* on the retina. The minimal area would then be 0.04 square *mm*. There are 13,500 cones per square *mm* of fovea, from which it appears that the minimal retinal are of 0.04 square *mm* contains 540 cones.

Finally there is the matter of color perception. If it is assumed with Thomas Young that there are three kinds of cones, red-sensitive, green-sensitive, and blue-sensitive, then the color of a light depends on the number of these three kinds of cones which is brought into play. We found the minimal retinal area to contain 540 cones. Granting three kinds of cones in approximately equal numbers, we get about 180 cones of each type per unit area. If, now, a spectral hue discrimination involves, on the average, a change in one of each species of cone, then the number of monochromatic patches obtainable with a spectrum should be about 180. It is significant that Koenig obtained 160 such patches; Jones and Nutting between 130 and 180; Laurens and Hamilton 161 and 207 respectively.

Summary.

1. Visual acuity varies in a definite manner with illumination, being low at low illuminations and high at high illuminations.

2. These variations in visual acuity measure the variations in the resolving power of the retina. The resolving power of a surface like the retina depends on the number of elements per unit area. Changes in visual acuity then presuppose that

the number of elements in the retina is variable. This cannot be true structurally; it is therefore assumed functionally.

3. It is assumed that the thresholds of the rods and the cones are distributed in relation to illumination in a statistical manner similar to other populations. A quantitative treatment of such a supposition is shown to describe the data of acuity accurately.

4. The visual acuity of completely color-blind eyes are shown to follow the deductions from this explanation.

5. Further analysis shows that color, form, and brightness vision may all be considered as properties of minimal retinal areas containing about 540 cones.

A TYPICAL FORM OF FAMILIAL DEGENERATION OF CORNEA (FLEISCHER).[1]

By Dr. C. H. CHOU.

(Department of Ophthalmology, Peking Union Medical College, Peking, China.)

(*With four illustrations on Text-Plate XXXIV.*)

IN 1905, Fleischer first described a very rare form of familial degeneration of the cornea. He found the disease in three members of one family, and described it as a process consisting of many small ring-shaped opacities together with many punctiform and nodular or flake-like opacities, occupying almost completely the central parts of both corneas. These opacities, differing from all other forms of familial degeneration, were deeply situated in the corneal stroma, extending nearly to Descemet's membrane. The corneal surface was even, and the corneal substance in between these nodules was clear. The condition had begun in early youth, and remained unchanged for quite a number of years.

In reviewing ophthalmic literature up to 1905, he was able to find two instances which might have some similarities to his cases. From 1905 up to the present year, I have found only two more families in all ophthalmic literature which were afflicted with this same disease. Before reporting our case, I will give an abstract of all the cases which have appeared in the literature.

Abstract.

I. Groenouw (2), in 1890, reported the case of a patient, Gottfried, T., aged 49, suffering from catarrh for eight days. Vision R. = $\frac{5}{60}$, L. = $\frac{5}{60}$. The right eye was pale; left eye

[1] Read before the 7th Biennial Conference of the National Medical Association of China, held in Peking, January 31, 1928.

ILLUSTRATING DR. C. H. CHOU'S ARTICLE, "A TYPICAL FORM OF FAMILIAL DEGENERATION OF CORNEA (FLEISCHER).

FIG. 1.—R. E.

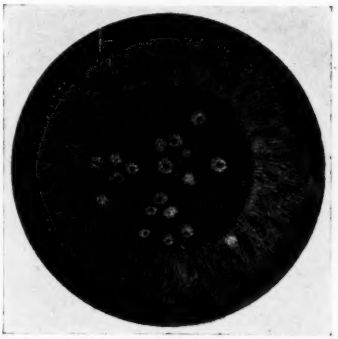


FIG. 2.—L. E.

Showing the general distribution of the rings and nodules of both corneas as seen under a loupe. They are situated in the pupillary areas. 20 of such opacities are found in the right eye and 19 in the left.

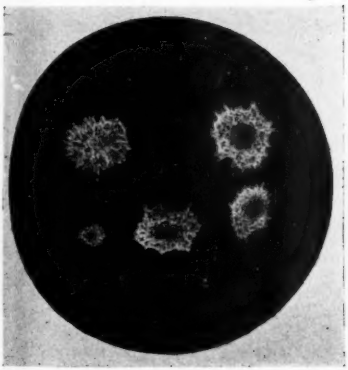


FIG. 3.

Oc. 3, Obj. A2.

The opacities are highly magnified as they were seen under corneal microscope. The opacities are discrete, and they are more or less indented with fine processes. The centers of the opacities are clear in some ring-shaped ones.

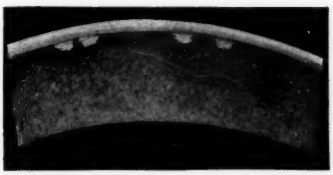


FIG. 4.

Oc. 3, Obj. A2.

A narrow optic section of the cornea, showing the localization of the opacities mostly underneath Bowman's membrane. A nerve filament is seen to pass thru the section.

showed marked catarrhal inflammation. In the central part of each cornea were many grayish round, and irregular opacities, with indentation and processes, described by Groenouw as "particles from dry bread." Some were discrete and some were arranged in groups or streaks. Between these opacities were fine punctated opacities. The periphery of the corneas was clear. The epithelium covering the opacities was slightly elevated. On examination of a removed piece, a hyaloid substance was found under Bowman's membrane, and the fine punctated opacities were considered by him simply as enlarged corpuscles.

II. Koerber (3), in 1902, reported the case of a patient, W. F., chemist, aged 28, who had had eye trouble since the age of 10. Vision R. E. = ⅓ with −1.00 sph. = ½ − ⅔, L. E. = ⅓ with −1.50 sph. = ½, Jaeger No. I at 17*cm.* The conjunctivæ were injected. The surface of cornea was slightly dull, and the epithelium slightly stippled. Sensibility was normal. In the central parts of the corneas there were rows of grayish, round, or oval, and indentated opacities, about ¼ to ½*mm* in diameter. Between them were seen numerous diffuse punctated opacities. The cornea looked clear where these opacities were absent. The edges of the larger opacities were more whitish than the centers, which in many places were clear.

III. Fleischer (1), in 1905, reported three cases (Cases XXXII–XXXIV).

(a) The first patient was a boy, G. B., 23 years of age. Vision R. E. = with −5.00 sph. −2.00 cyl. × 90 axis = $\frac{5}{6}$ −$\frac{5}{6}$, L. E. = with −4.00 sph. −0.75 cyl. × 90 axis = $\frac{5}{6}$ −$\frac{5}{6}$.

In the deeper layers of the central parts of the corneas were found many small, round, and irregularly outlined flakes; some of them appeared as little rings because the centers were quite clear. The corneal substances between the opacities were clear. The epithelial surface was smooth. Eye, not inflamed. Fundi, normal.

(b) H. B., brother of G. B., aged 19.
Vision R. E. = with −2.50 sph. = $\frac{5}{6}$, L. E. = with −4.20 sph. = $\frac{5}{6}$ − $\frac{5}{6}$.
The corneal picture was similar to that of his brother.

(c) The mother of G. B. and H. B. was examined when she was forty-four.
Vision R. and L. = $\frac{5}{80}$ with + 0.50 sph. = $\frac{5}{10}$ − $\frac{5}{15}$; +3.00 sph. read Jaeger 1.

In the central part of each cornea were found many little ring opacities, some of which were not complete.

IV. Hancock (4) reported two cases in 1905.

(a) Mrs. G., aged 45. Her sight was poor, and slowly became worse.
Vision R. = $\frac{5}{60}$, with +3.00 sph. = $\frac{5}{15}$; with +4.00 sph. Jg. 4 badly.
L = $\frac{5}{60}$, with +3.00 sph. = $\frac{5}{15}$; with +4.00 sph. Jg. 4 badly.

In the central part of each cornea, was a creamy white opacity, roughly circular in shape, leaving the periphery of the cornea perfectly clear. The opacity was made up of closely-set, rounded, opaque areas. In the lower part, these areas had a distinct tendency to form rings with clear cornea within the little circles. The epithelium was partly raised. No anesthesia. Tension normal.

(b) H. G., aged 11, the son of the first patient.
Vision R. E. = with +6.00 sph. = $\frac{5}{12}$ and Jaeger 1 for near.
L. E. = with +6.00 sph. with +1.00 cyl. × 90 axis = $\frac{5}{8}$ and Jg. 1 for near.

Corneal condition similar to that of his mother, but much less, and more discrete, with a greater tendency to ring formation. The corneal surface was smooth. No anesthesia.

V. Hine (5), in 1923, reported the cases of a mother and her son and daughter.

(a) Mrs. B., aged 36. Her eyes had troubled her since infancy.
Vision R. = $\frac{5}{18}$ with +1.00 sph. +0.50 cyl. × 90 = $\frac{5}{15}$.
L. = $\frac{5}{12}$ with +1.00 sph. +0.50 cyl. × 90 = $\frac{5}{12}$.

The central part of the corneas was studded with numerous close-set, grayish-white opacities, some denser than others;

some in the form of small rings, and others nodular in shape. Periphery of the cornea was clear. No general haziness.

(b) G. B., boy, aged 14.
Vision R. = $\frac{6}{6}$ with +2.50 sph. = $\frac{5}{5}$
L. = $\frac{6}{12}$ with +2.50 sph. +0.75 cyl. × 90 = $\frac{5}{6}$

Corneal picture similar to that of his mother, but there were more rings, and fewer nodules. The apparently clear cornea between the opacities showed some almost invisible greyish specks under the epithelium, which the author considered to be due to an impregnation of a fine translucent granular deposit which may be a crystalline substance having a refractive index differing from that of the cornea. The bigger opacities might be a condensation of these deposits.

(c) Lily G., aged 10.
Vision R. = $\frac{6}{12}$ with +2.50 sph. +0.50 cyl. × 90 = $\frac{5}{6}$
L. = $\frac{6}{6}$

The opacities of the right cornea were composed of fine lines, which did not form a meshwork. The left cornea showed a picture of the combination of two linear opacities with several small rings. In all these cases, the anterior surface of the opacities lying in a plane with Bowman's membrane.

The report of the case which we have seen recently will be made in full including slit-lamp examination.

Hosp. No. 18991, Miss C. F. S., age 27, a school teacher, came to the Peking Union Medical College Hospital in December, 1926, complaining of eyestrain after prolonged use. Vision was $\frac{6}{15}$ plus 1 for the right eye, and $\frac{6}{12}$ plus 2 for the left. Conjunctiva slightly congested. No notes about the cornea. Pupils normal. Fundi normal. Refraction was advised, but the patient did not return to the clinic until Septemer 22 of 1927, when she complained of blurring vision. Fundi remained unchanged.

In the central part of each cornea were numerous small greyish, roundish, flakelike opacities, most of which were more or less irregular in outline with fine indented processes. There were twenty such nodules in the right eye and nineteen in the left. Most of these flakes had clear centers, and thus appeared in the form of rings (Figs. 1 and 2). The corneal surface was smooth, and lustrous.

Slit-lamp Examination:

R. E. The big nodules appeared as rather irregular outlined opacities, as if they were formed of numerous fine particles, arranged in a ring-shaped fashion. The periphery was more saturated, so appeared to be more opaque than the central area (Fig. 3). Between these ring-shaped nodules were also seen numerous fine, not saturated particles. They were more distinct in the indirect image as fine dew drop-like, glassy opacities than by the direct illumination. In the central part of the cornea, the nerves appeared varying in whiteness, and were visible even to the smallest end branches. They were not often obscured by the denser greyish parts of the cornea. They seemed to have no connection with these opacities, though occasionally some of such nodules were found along the nerve filaments. Around some of these nodules where no visible opacities were seen, the stroma was filled with many opaque, unsharply defined microscopical fine particles, among which the nerves were more easily visible than in the normal cornea. The anterior half of the cornea showed marble condition. The deeper layers of the corneal lamellæ were normal.

The endothelial layer was normal in the periphery. In the central region of the cornea, the posterior endothelial mosaic became enlarged, roughened, and showed glistening reflexes (the beginning of the degeneration of endothelium).

All the opacities were lying behind the epithelium, some of them in Bowman's membrane, but most of them in the superficial lamellæ of the corneal stroma, about its anterior $\frac{1}{8}$ of the cornea (Fig. 4), and apparently situated in different planes.

Some floating opacities were found in the anterior chamber, and some fine whitish corpuscular elements were seen on the anterior lens capsule, besides the congenital brown star cells visible on the temporal upper quadrant. Iris normal.

L. E. The condition of the left eye was similar to that of the right. A transparent glassy line was found between and a little behind two of the bigger nodules. Some of the nerves also appeared as glassy, long straight filaments in the indirect illumination. Some dew drop opacities were present in the posterior surface of the cornea.

Tension—On September 24, 1927.

R. E. 10
L. E. 8 (Sch.)

On March 23, 1928.
R. E. and L. E. = 16 (Sch.)

The corneal sensibility was somewhat reduced.

In the family history, there is no consanguinity. Parents are living and well. Their vision are said to have become poor recently (probably presbyopia), but there are no similar corneal opacities noticed. One brother with perfect vision is living and well. There is no evidence of similar trouble among her near relatives.

General physical examination revealed a rather obese female adult. A soft systolic functional murmur was heard at the apex. Slight tenderness over right hypochondrial region. No evidence of local degeneration in any other part of the body. Urine, feces, and blood examinations were all normal. A basal metabolic test was made and found to be within the normal limit. Wassermann and Mantoux tests were all negative.

About three months later on further examination, there seemed to have been some increase in the fine dust-like opacities, while the central large ones remained unchanged.

During the past months, the patient has been using yellow oxide of mercury, massage and dionin. Radium at 15*mgm* hours was given at fortnightly intervals to alternate eyes.

Comment.

The above reported cases in the literature together with the present case constitute a distinct form of the group of the disease known as "familial degeneration of the cornea." The familial degeneration of the cornea, as has been described in the literature, may occur in various forms and shapes. The picture may be, therefore, predominated by the formation of a meshwork, nodules, fine punctated opacities, small flake-like, disc-like or sausage-shaped opacities. The ring-shaped form which may be just another form of the same disease, has many points which are commonly characteristic of all other forms. This will be further supported by Hine's case V (c), where in the right eye he found numerous lines, while in the left eye he found a combination of two lines and many small rings.

The salient characters of this disease, according to Fleischer, are the ring-shaped opacities, the evenness of the surface, the clear corneal substance in between the opacities. All these points are present in our case. According to Fleischer, the opacities were all deeply situated, but the slit-lamp examination reveals them to us apparently only in the anterior ⅛ of

the corneal stroma, just beneath Bowman's membrane, as also in Hine's cases. The exact localization of the opacities is quite impossible without the help of the slit-lamp. In Fleischer's cases, the opacities, which he found were situated in the deeper layers of the corneal stroma, would most probably have been seen in the superficial lamellæ had the cases been examined with slit-lamp.

The progress in this form of the disease seems to be particularly slow, and the prognosis as recorded in the literature for useful sight is very much better than any other form. In Fleischer's patient, aged 47, the vision with full correction was $\frac{5}{10}$ and $\frac{5}{7.5}$. Hancock's patient had vision of $\frac{5}{15}$ at age of 45, and Hine's patient, aged 36, had vision of $\frac{5}{15}$ and $\frac{5}{12}$. In Groenouw's case the vision was reduced to $\frac{3}{60}$ and $\frac{5}{60}$ at the age of 49. In most other forms of the disease, so far as recorded, the vision would have been reduced to finger count when the patient reached middle age. On the other hand the present case has not been observed long enough to say definitely what the state of progress will be in more advanced years.

With regard to the real nature of the disease, the etiology, as in any other forms of the degeneration, is still very dark, all the theories being on a presumptuous basis, and the present case will probably not throw any light on the subject. Pillat (6) who found in his case of mixed nodular and reticular degeneration, the enlargement of the nerve endings and great unequal thickening of the nerve filaments, has the opinion that the corneal degeneration is primarily a nerve disease, and that around the diseased nerves there is a trophic neurotic degeneration of the corneal lamellæ. Pascheff (7), who also found the glassy lines by indirect illumination, has the opinion that the corneal corpuscles first became opacified and later follows the degeneration of the corneal nerves. On the other hand, A. Fuchs (8) is of the opinion that the corneal nerves are not diseased. That the unequal thickening might be due to the presence of glial tissue in unequal amount in various places along the same nerve, and that the slight reduction in the sensibility does not indicate affection of the nerves. It is not uncommon to find slight reduction in sensibility in any other chronic inflammatory conditions of the eye. He has observed

furthermore a corneal ulcer with hypopyon developing in a case of familial degeneration of the cornea, and both the ulcer and hypopyon disappeared in a short time after being treated. He has the opinion that the disease is primarily a degeneration of the corneal lamellæ.

It has been a known fact that all the cases developed first in the central part of the cornea, and that a zone of periphery clear cornea is always present even in well advanced cases. The central part of the cornea, where the nutrition is the poorest, suffers first whenever there is any disturbance in nutrition. Any low grade local metabolic disturbance of some obscure etiology may set up such a change, as the degeneration of the connective tissue concerned, with the deposition of such substance as hyalin. This local metabolic change may perhaps be due either to a degenerative change in the peripheral nerve endings or perhaps some endocrine disturbance.

Macrae (9) reported some cases of nodular keratitis in Arabia, a few of which had also ring-shaped forms, from which he made the presumption that in his series of cases, it might be the result of the impregnation of salt particles. His cases may not belong to the same disease, as he found they were usually associated with leucoma and ulceration, some of them being also unilateral.

Heredity certainly plays an important rôle. All the cases except Groenouw's and Koerber's occurred in more than one member of a family. Although we were not able to examine any other members of our patient's family, nevertheless we still have to consider the present case as a familial condition, as it has all the clinical salient characteristics of the disease. If our patient is the only one in her family afflicted thus far, she may be the first, and later we may find the similar condition in her descendants.

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TUBERCULIN THERAPY IN OCULAR TUBERCULOSIS.

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FOR many years it has been known that tuberculosis of the eye responds favorably to subcutaneous injections of tuberculin. In recent years the diagnosis of ocular tuberculosis has been made much more frequently, chiefly because the oculists have been on the lookout for such infection in chronic cases, and because of the introduction of the slit-lamp and the wider use of specific serologic reactions. The internist is being called on much more frequently to assist in establishing a positive diagnosis in chronic inflammations of the eye. This entails not only a careful physical examination, the frequent use of the X-ray for diagnostic purposes, but also the use of the numerous examinations of the blood and immunologic reactions.

Tubercular reactions similate syphilitic reactions, and this fact is readily demonstrated in the eye where the lesions can be examined directly. Through the researches chiefly of Ghon, Lubarsch and Ranke, we know that the primary seat of infection is the respiratory tract, although in children the entrance point may be the intestinal tract in from fifteen to twenty per cent. of cases. The primary changes may be microscopic only and are characterized by the stimulation of the fixed connective tissue leading to the production of epitheloid cells through the action of the tubercle bacillus and its toxines. The blood vessels become involved leading to degenerative changes in the tubercle which has been formed, frequently with the production of giant cells. The process may stop at this point or the surrounding tissue becomes involved resulting in infiltration with lymphocytes, œdema and hyperemia, which may be looked upon as a perifocal

reaction with a tendency to the isolation of the tubercle. Immunologically speaking, this primary stage may be considered as the stage of sensitization to the tubercle toxin and is characterized by proliferative and marked connective tissue changes.

Through trauma or disturbed circulation, the secondary stage may arise which is the period of generalization, marked by local and general reactions involving distant organs by metastasis through the blood or lymph stream, which may be likened to chronic bacteriemia causing a state of hypersensitiveness or anaphylaxis. The symptoms may be violent or mild depending upon the resistance of the individual and immunologic response which has been aroused. In the tertiary stage the inflammatory process is less marked, is more abortive in nature, and the stage of immunity has been reached. In the eye, the lid or conjunctiva may be the seat of a primary infection from without, although this is very rare. The infection gains entrance to the eye through the circulation and is most frequently a metastatic process. Schick first recognized the primary stage in the small nodule occurring in the iris with a slight area of resorption; the secondary stage in the diffuse iritis and irido-cyclitis with acute toxic symptoms; the tertiary stage with the torpid iritis and later formation of glass-like nodules and precipitates. In tuberculosis of the eye we may have an acute process with symptoms of hypersensitiveness and a tendency to destruction; such a patient should be treated by rest and general measures, rather than through the use of specific medication. Again we may have a proliferative type without acute manifestations and with a tendency to connective tissue formation; patients in this stage are frequently benefited by specific medication. The tubercular infection need not necessarily follow all three stages and the second stage in particular need not necessarily be severe and may be without marked symptoms of hypersensitiveness.

General Symptoms: As a rule these are not marked in ocular tuberculosis as we meet with it in general practice. It is because of this that the diagnosis is usually not made. Although commonly a metastatic process, the primary lesion is hidden somewhere in the body, usually in the bronchial or

peribronchial lymph glands. Physical and X-ray examination may detect a latent process but the clinician hesitates in pronouncing this focus as the cause of a given ocular infection. Finnoff (1) has removed several eyes in which a typical tubercular process could be demonstrated, the patients showing no clinical or Roentgen-ray evidence of the disease elsewhere. Anemia, loss of weight, slight evening elevation of temperature and asthenia may be present, symptoms which are so commonly met with in daily practice. Therefore a thorough physical examination is imperative and the findings should be considered especially from the standpoint as to whether they may possibly be causing a given ocular infection.

Rollet and Colrat (2) state that tuberculosis of the uveal tract is always of secondary origin and hold that the primary focus of infection is situated in the peribronchial lymph nodes. In eighty per cent. of one hundred cases they were able to demonstrate Roentgen-ray findings in the chest, these changes consisting of definite shadows at the base of a lobe, usually the lower, surrounded by healthy parenchyma, with peribronchial striæ connecting the shadow with the hilus gland shadow. They object to the use of tuberculin because of the tendency to fever reactions and the difficulty of correct dosage. They prefer employing the X-ray as a therapeutic measure and, with certain precautions, radio therapy.

Specific Reactions: Although these may be elicited in any patient who has previously been infected with the tubercle bacillus, the detection of such a reaction may be of great value in establishing a diagnosis of ocular tuberculosis. The negative result is frequently of particular value because patients suffering from ocular tuberculosis are prone to develop a focal reaction, particularly when the subcutaneous method of injection has been employed. The Wassermann reaction for lues should always be made, because of the similarity of tubercular and syphilitic processes. A distinctly positive Wassermann reaction should lead to the employment of antisyphilitic measures, even in the presence of specific tuberculin reactions. The simple cutaneous or Von Pirquet test is of value chiefly when negative; the positive result should lead to the use of the intradermic or subcutaneous method.

Mantoux (3) introduced the intracutaneous test, injecting 0.1 to 0.05*cc* of a 1:5,000 solution of O. T. In the majority of the cases here listed the stick reaction was employed; a drop of the old tuberculin was placed upon the sterilized arm, and a sterilized darning needle was used to introduce the tuberculin intracutaneously. The degree of reaction was noted at the end of 24 and 48 hours. In cases numbered 1, 11, 13, 15, and 16, the tuberculin was introduced intracutaneously with a fine needle attached to a tuberculin glass syringe. Five dilutions, 1 to 1,000,000, 1 to 100,000, 1 to 10,000, 1 to 1,000, and 1 to 100 were employed. Normal individuals may react to 0.05*cc* of a 1 to 1,000 dilution, therefore, reactions occurring with higher dilutions indicate the presence of clinically active tuberculosis. By using 0.1*cc* of each dilution a total of 0.0011111*cc* of old tuberculin will be introduced, an amount sufficient to produce a mild focal and general reaction. When such reactions are to be avoided, it is more advisable to use the Mantoux technique. To avoid severe focal and general reactions it was not deemed advisable to use the subcutaneous method as a routine. Pain is usually absent in ocular tuberculosis, but when present is frequently relieved by slight focal reactions. The temperature should be taken throughout the day and any elevation noted. The so-called "tubercumet reaction" which is dependent upon the presence of precipitins in the patient's blood detected by allowing a preparation of the tubercle bacillus as antigen to come into contact with an equal amount of patient's clear serum, was not found to be of any great value in establishing a positive diagnosis because it is frequently positive when no other findings would warrant the diagnosis of clinically active tuberculosis.

Abstracts of Case Histories.

Case I.—Mrs. L. L., aged 31. Clinical diagnosis: acute uveitis, beginning in right eye with injection, floating opacities, no pain, no headache. Later, optic neuritis with hyalitis developed. Because of a positive intradermic tuberculin reaction using a dilution as high as 1–100,000 O. T., specific therapy was begun, using $\frac{1}{500}$ *mgm* T. R. Only a few injections were given with no benefit. Glau-

coma developed for which a punch operation was performed to relieve pressure. Seven more injections were given but with no benefit; subsequently it became necessary to remove the right eye. Three years later, she complained of vague symptoms in the left eye with floating opacities. Tuberculin injections were given by another physician but with no benefit. Result: tuberculin injections were of no benefit.

Case II.—Miss J. S., aged 40. Clinical diagnosis: tuberculous scleritis. Family history of tuberculosis. Patient likewise treated for tubercular hip as a child. History of similar eye involvement in the past. Present attack associated with headache and some pain in the eye. Marked local, some focal and general reaction following the intradermic test using the stick reaction and concentrated old tuberculin. Tuberculin therapy using T. R. and B. E. has been given at intervals over a period of nearly four years, as much as $\frac{1}{10}$*mgm* being used at one time. Result: marked improvement, relief of all pains, improved vision, is able to work as a dressmaker.

Case III.—Miss N. H., aged 35. Clinical diagnosis: tuberculous irido-cyclitis, involving chiefly left eye, associated with pain and headache. Family history of tuberculosis and patient gives a history of previous pulmonary tuberculosis. Local and general reactions following the intradermic test using the stick reaction and concentrated old tuberculin. Result: marked clinical improvement with relief of pains following tuberculin therapy, using subcutaneous injections of T. R., beginning with 1 to 1,000 and ending with $\frac{1}{10}$*mgm* over a period of seven months.

Case IV.—Miss J. S., aged 45. Clinical diagnosis: chorioretinitis centralis, bilateral. No family history of tuberculosis. Patient has had pulmonary tuberculosis in the past. Local reactions to the intradermic tuberculin test using the stick reaction and concentrated old tuberculin. It was thought unwise to use the subcutaneous method. Injections of tuberculin given elsewhere resulted in some clinical improvement. Result: eye condition improved.

Case V.—Mr. J. R., aged 28. Clinical diagnosis of tuberculous iritis was made with the means of the slit-lamp. No family or personal history of tuberculosis. Intradermic tuberculin test using the stick reaction and concentrated old tuberculin was positive; subcutaneous method not used. Subcutaneous injections of T. R., sixteen in all, the last one

employing $\frac{1}{10}$*mgm* were given. At no time was there any elevation in temperature and no general symptoms; local reactions always marked. Result: no improvement which could be attributed to the use of tuberculin.

Case VI.—Mrs. N. S., aged 39. Clinical diagnosis: tuberculous uveitis. Family and personal history negative for tuberculosis. Distinct attacks of cholecystitis in the past. Right eye injured while gathering cobs in the field two years previously; removed two months ago. Now the left eye is becoming involved, uveitis developing. The optic nerve was normal, fundus normal, vision $\frac{1}{10}$, low grade uveitis with slight scleritis. No evidence of tubercular involvement of the lung. Intradermic tests using the stick reaction and concentrated bovine and human old tuberculin were strongly positive. Seventeen injections of tuberculin T. R., mixed varieties given over a period of five weeks with a maximum dosage of $\frac{1}{10}$*mgm* T. R. human and $\frac{6}{10.000}$*mgm* bovine, were given. Three months later the eye was apparently cured of all active trouble; there remains the lens opacification above the pupil space and the old punctate depositis, now scars, no Descemets' membrane. Result: very satisfactory.

Case VII.—Mr. J. C. M., aged 29. Clinical diagnosis: retinal hemorrhage, more recent episcleritis of each eye. No family or personal history of tuberculosis; no tubercular changes in the lung detected. Intradermic test using the stick reaction and concentrated old tuberculin, was strongly positive. Infected tonsils were removed and a pathologic examination showed them to be tubercular. Two weeks later tuberculin injections were begun using T. R. and B. E. Thirty-five injections over a period of five months were given, with a maximum dosage of $\frac{1}{10}$*mgm* T. R. and $\frac{1}{10}$*mgm* B. E. Two months later the oculist reported that the eyes had healed entirely. Result: very satisfactory.

Case VIII.—Miss A. E. D., aged 35. Clinical diagnosis: tuberculous iritis. Had an attack of interstitial keratitis in same eye formerly; this cleared under general treatment. History of tubercular glands of the neck. Episcleritis developed in the same eye and two days later an acute attack of iritis with nodules typical of tuberculosis. Intradermic tuberculin test using the stick reaction and concentrated old tuberculin was strongly positive with papule and blister formation. Twenty-six injections of tuberculin T. R. and B. E., over a period of eleven months were given;

the local reactions were always marked, but no focal or general reactions occurred. The maximum dosage was $\frac{1}{10,000}$*mgm* B. E. and $\frac{1}{10,000}$*mgm* T. R. Result: distinct clinical improvement; the iris healed completely.

Case IX.—Mrs. H. W. McK., aged 57. Clinical diagnosis: iritis, recurring tubercular infection of left eye, obesity and hypertension. Two distinct nodules in left eye in the iris at pupillary border; gelatinous like floating particles in vitreous. Intradermic tuberculin test using the stick reaction and concentrated old tuberculin was strongly positive. The acute irido-cyclitis was at first treated with hot packs and subconjunctival injections. Later subcutaneous injections of tuberculin T. R. were given over a period of six months. The maximum dosage was $\frac{1}{100}$*mgm* T. R. Result: improvement was marked; the affected eye free from inflammation. Vision $\frac{1}{2}\frac{8}{0}$.

Case X.—Mr. J. F. C., aged 20. Clinical diagnosis: tuberculous iritis in each eye, just beginning in the left. The intradermic tuberculin test using the stick reaction and concentrated old tuberculin was strongly positive. Twenty injections of B. E. and T. R. over a period of four months resulted in marked improvement. The maximum dosage was $\frac{1}{100}$*mgm* B. E. and $\frac{1}{100}$*mgm* T. R. Patient was seen again eight months later and reported that he had been fine, that the eyes had been in good shape, the left better than the right. He had gained five pounds in weight. Result: marked improvement.

Case XI.—Mr. G. W. J., aged 30. Clinical diagnosis: tuberculous choroiditis, left eye. Intradermic tuberculin test was strongly positive with a dilution of 1 to 1,000,000 old tuberculin. Patient was given Tebeprotin (Toenniessen), eleven injections in all, the last one—1*cc* of dilution 2. Result: some improvement; the patient then ceased coming.

Case XII.—Miss M. W., aged 21. Clinical diagnosis: destructive choroiditis in right eye since childhood; now in left eye there are opacities in the vitreous and irido-cyclitis is present. Although the patient had a negative Wassermann test, owing to the history of a positive test in the father, preliminary mercury injections were given with no benefit. Intradermic tuberculin tests were negative, using old tuberculin. Tebeprotin injections were given; local reactions were only obtained when the maximum dose $\frac{1}{10}$*mgm* was employed. This was repeated nine times over a period of two months. There was some improvement, the eye beginning to clear, the scar becoming thinner.

Case XIII.—Mr. T. B., aged 41. Clinical diagnosis: double keratitis and uveitis. The attacks began eighteen years ago with uveitis and exudates probably tubercular in origin. One sister died of tuberculosis. Owing to the marked clouding of the cornea, the patient had difficulty in getting about. The intradermic tuberculin reactions using old tuberculin were distinctly positive in a dilution as high as 1 to 10,000, and were accompanied by a general reaction which was followed by a clearing of the cornea so that the patient was able to get about much better. Another subcutaneous injection of $\frac{1}{10}$*mgm* old tuberculin was again followed by local and general reaction and further clearing of the cornea, so that the patient could walk by himself.

Case XIV.—Mr. C. G. M., aged 35. Clinical diagnosis: old choroiditis disseminate in both eyes, with recrudescence in the left eye. No family or personal history of tuberculosis. The intradermic test using the stick reaction and concentrated old tuberculin was only slightly positive. There was a slight reaction in the eye leading to iritis and cyclitis, but this gradually subsided under treatment. Subcutaneous injections of tuberculin T. R. and B. E. given over a period of two years, sixty-four in all. After the early injections, some general reaction occurred with some elevation in temperature. A single focal reaction cleared quickly. He previously had treated with the same oculist for two years with no benefit. The choroiditis has greatly improved, no evidence of hemorrhage or exudation, but some scar formation leading to imperfect vision. Result: distinct improvement. During the first year's treatment, his weight increased from 173 to 188 pounds.

Case XV.—Mrs. A. B., aged 32. Clinical diagnosis: tuberculous irido-cyclitis in left eye of three months standing. The intradermic tuberculin reactions were strongly positive with the higher dilutions and were followed by general reaction. Cervical lymph glands become smaller under treatment. Her brother had similar glands removed and these were found to be tubercular. Patient was given tebeprotin beginning with 0.3*cc* of dilution 3. Local reactions always occurred. She previously had headache and this was quickly diminished under treatment. Eighteen injections of tebeprotin were given over a period of three months. The maximum dosage was $\frac{1}{10}$*mgm*. The weight increased from 131 to 144 rounds during the first two months. Although the eye has not cleared completely,

there being still some evidence of iritis, the backgrounds are normal, no nodules are present, and she sees as well as with the other eye. Result: marked improvement.

Case XVI.—Mr. J. D., aged 49. Diagnosis: tuberculous irido-cyclitis of right eye. Attacks have been coming at intervals for the past five years. Intradermic tuberculin reactions using old tuberculin were strongly positive with a dilution as high as 1 to 1,000 and were accompanied by a focal reaction in the eye. Subcutaneous injections of tebeprotin were given, each one accompanied by local reactions. The maximum dosage was $\frac{1}{10}$*mgm.* There has been a rapid improvement, the patient returning to work as a painter within three weeks time. Twelve injections were given over a period of four weeks.

Case XVII.—Mr. W. H. S., aged 35. Clinical diagnosis: retinal hemorrhages of at least six months standing. The oculist reported a hole forming process in the choroid near macula lutea of right eye; vision reduced to $\frac{4}{10}$. Process was still active with a hemorrhage still visible on the temple side. The intradermic reactions using old tuberculin were slight but with larger doses of tebeprotin, it became more marked. No focal or general reactions developed. Sixteen injections of tebeprotin were given subcutaneously, including three injections of the maximum dose of $\frac{1}{10}$*mgm* over a period of three months. At the end of this time, the oculist reported no signs of activity. The small hemorrhage which was discovered in the back of the right eye had entirely disappeared.

Discussion: In ten of the seventeen cases, both general and ocular improvement quickly followed the use of tuberculin given subcutaneously. These were cases 2, 3, 6, 7, 8, 9, 10, 14, 15, and 16. The patients were satisfied and were able to resume their occupations. In case 10, the keratitis improved following two injections of old tuberculin, sufficient time not having elapsed for further observation and treatment. Case 1 showed no improvement and the conclusion was reached that the ocular condition was not due to tubercular infection. The remaining five showed some improvement, four of these being accompanied by choroiditis. In case 17, the retinal hemorrhage and surrounding inflammation disappeared entirely under treatment.

The patients who reacted in a general way by some elevation in temperature and an increased reaction in the eye, responded quickly, and the subsequent improvement in the ocular condition was very marked. In no instance did any harm follow such mild reactions. All of the patients realized the seriousness of their conditions, cooperated fully, and were free to express their gratitude when the local condition had improved. Since there was no other evidence of an active tubercular process, it was not necessary to introduce other general therapeutic measures and the favorable results were readily ascribed to the tuberculin therapy. Most of them were treated with tuberculin B. E. and T. R., and there was no need of changing the preparation for another. Four of the patients received injections of Tebeprotin, prepared after the method of Toenniessen. This is a purified water soluble preparation of the bacterial cell bodies. Following its use the local reactions of the skin were frequently marked, but such reactions always subsided. It was aimed to reach the maximum dose of $\frac{1}{10}$*mgm* as quickly as possible, but frequently a smaller dose was repeated to avoid reactions.

Summary.

1. Tubercular infections of the eye are amenable to tuberculin therapy.

2. Although it was not aimed to secure general or focal reactions, when they did occur, improvement in the ocular condition usually followed.

3. Tuberculin B. E. and T. R. and the new preparation "Tebeprotin" of Toenniessen were employed and found very satisfactory.

4. Diagnostic tuberculin tests are of value in establishing the proper diagnosis, particularly when accompanied by mild general and focal reactions.

5. When ocular tuberculosis is accompanied by symptoms of tuberculosis in other organs, general measures should first be employed.

6. In long standing or recurring infections of the eye, physicians will do well to think of the possibility of an underlying tubercular infection, and make the necessary general and special examinations.

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Note: The writer wishes to express his indebtedness to the oculists, Drs. Robert Sattler, D. T. Vail, Jr. and Sr., and Clarence King, who made this study possible.

ON THE OPERATION FOR SHRUNKEN CATARACTS IN ADOLESCENTS.[1]

By Dr. ARNOLD KNAPP, New York.

IN young people there is a form of cataract which is shrunken and often calcareous, and represents the final stage of a congenital cataract. It usually affects only one eye, there are no adhesions of the iris, and the chief objection is its cosmetic blemish from which the patient is very anxious to be relieved. The usual methods of operating do not hold any hope of relief, for these cataracts cannot be needled or extracted in the usual way, while even if they can be cut through by a knife-needle or scissors, the wound does not gape and the deformity remains in the pupil. Total extraction by grasping the cataract with the capsule forceps is possible if the cataract can be seized, and there are no adhesions to the ciliary processes. The operation necessarily must be done with the preservation of a round pupil for cosmetic reasons. Usually the cataract is so shrunken and flat that it cannot be seized with the capsule forceps and extracted, and adhesions to the ciliary processes contraindicate unavoidable traction. Then the following procedure can be recommended. This method was successfully practised in the two cases which form the basis of this paper.

The pupil is first fully dilated to determine the presence, location and extent of any adhesion to the ciliary processes. A keratome incision is then made just within the limbus where the periphery of the pupil is clear, adjoining the thickened peripheric capsular adhesion. The keratome after

[1] Read at meeting of Am. Ophth. Soc., Washington, D.C., April 30, 1928.

passing through the cornea, perforates the clear capsule and the opening in the capsule is enlarged by lateral incisions. This causes the pupil to remain dilated by prolapse of vitreous into the anterior chamber, and facilitates future steps. With the de Wecker capsule scissors (Esberg's model) the peripheric capsular adhesion is then divided. The shrunken cataract is seized with the blunt capsule forceps (Kalt) or with the Liebreich forceps modified by Panas, the sharp blade introduced in the vitreous behind the cataract and with the blunt blade on its anterior surface, the cataract is grasped and cautiously drawn out of the eye. A bead of vitreous may present at the corneal incision; it is cut off with the scissors applied on flat. The result is a black, round pupil, and a satisfactory cosmetic result is obtained.

The cases are the following:

(1)—G. S., aged 14, congenital cataract with broad thickened adhesion nasally. The pupil dilated fully. Local anesthesia. Keratome incision in lower cornea entered the clear pupil below the cataract. The capsule scissors divided the broad capsular adhesion by a vertical cut, at the nasal margin. Vitreous presented. The Panas forceps then seized the cataract and the cataractous mass was freed and cautiously pulled out. Slight amount of cortex remained. Vitreous snipped off. Atropin salve, bandage. Healing without incident, cortex absorbed, black pupil.

(2)—F. P., aged 26, congenital cataract, now shrunken, with broad capsular adhesion above and nasally. Pupil reacted promptly. As there was some clear cortex above and nasally, a discission was first done in the upper and then nasal periphery. The resulting free cortex in the anterior chamber was evacuated 10 days later. Two months later the extraction was done under local anesthesia as follows: keratome incision down and in through clear pupil. The capsule scissors then cut through adhesion above. The cataract was seized with the Kalt forceps, and cautiously pulled out. Bead of prolapsed vitreous snipped off. Healing prompt. Round, black pupil.

The main point in this operation consists in cutting the broad capsular adhesion before extracting the cataract. Thus, traction on the ciliary processes was avoided. When the

adhesion is divided, the cataract can be grasped and extracted without trauma to the eye, and a round black pupil is obtained. This method is simpler and safer, to my mind, than the various operations which have been suggested for this form of shrunken cataract.

CORRECTION OF DEFECT DUE TO THIRD NERVE PARALYSIS.[1]

By Dr. MEYER WIENER, St. Louis, Mo.

PARALYSIS of the third nerve, partial or complete, leaves a very ugly and embarrassing disfigurement. This is all the more deplorable when the paralysis is permanent. Heretofore, patients so afflicted were given no encouragement. If operative means were resorted to, no permanent relief could be expected.

The only ray of hope offered before the present time was a suggestion made by Edward Jackson, and independently by Dransart that the transference of the tendon of the superior oblique to take the place of the paralysed internus might help relieve the defect. Jackson (1) recommended, for paralysis of the oculo-motor nerve, that the superior oblique be taken up, the tendon isolated, and divided far enough from the eyeball to furnish a sufficient length of tendon, the pulley then cut, the tendon then drawn down and toward the eyeball, and the cut end of the tendon sutured to the paralysed internus near its insertion. This operation had been done by Jackson on the cadaver but not on the living patient. He suggests that perhaps even a little better movement may be secured by subsequently splitting the external rectus for 15*mm* back from its insertion, and transplanting the upper portion to the temporal edge of the insertion of the superior rectus and the lower portion to a corresponding relation with the inferior rectus. It is possible that in a young person a limited field of binocular fixation and stereoscopic vision could thus be obtained.

[1] Read before the Sioux Valley Eye, Ear, Nose and Throat Academy, Omaha, February 15, 1928.

Transference of the tendon of the superior oblique to the external rectus has been reported by Dransart (2) who also proposes the grafting of the tendon upon the upper edge of the externus, or the nasal edge of the inferior rectus. He suggests an incision through the skin at the upper inner angle of the orbit to expose the oblique at the pulley, and tunneling from there to the new point of attachment and drawing the tendon into the new position by a suture.

Neither of these authors had ever tried the operation on the living patient. I was not cognizant of either of these articles when the idea first occurred to me. I also had a simplified technic in mind, the practicability of which has been proved.

It may be well to refresh our minds with a brief review of the anatomical relationship. According to Whitnall (3) the superior oblique muscle lies along the angle between the medial wall and roof of the orbit. It arises by a short, slender tendon, anterior and medial to the optic foramen, from which it is separated by the medial rectus, in the angle between the annulus of Zinn and the periorbita. It may further be attached to the medial orbital wall for a few *mm* from its origin. The belly of the muscle, more rounded than that of the recti, runs forward, parallel and close to the nasal wall above the medial rectus, but separated from the latter by the nasociliary nerve and ophthalmic artery, and crossed by their ethmoidal branches; a few *mm* behind the orbital margin at its supero-medial angle, the muscle becomes tendinous and passes through a cartilaginous ring, the pulley or *trochlea:* it then bends abruptly backward, slightly down, and lateral-wards at a "trochlear angle" of about 54 degrees, and the round tendon broadens out, pierces the fascia bulbi, and passes beneath the superior rectus, to be inserted into the back and lateral side of the eyeball. The first part of the muscle from its origin to the trochlea is referred to as the direct portion, and the tendinous part between the trochlea and the globe, as the reflected portion. The tendon measures 20*mm* or ¾ inch in length from pulley to globe and is enveloped by a prolongation of the fascia bulbi, as far as the pulley.

The line of insertion of the superior oblique tendon crosses the vertical meridian obliquely, and is curved with a backwardly directed convexity; the breadth as measured by Fuchs

averaged 10.7*mm*. The distance from the corneal margin to where the line crosses the meridian is from 16. to 17.9*mm*. (Fuchs.)

The trochlear, or fourth nerve crosses over the muscle from the mesial side to enter its substance at about 12*mm* from its origin; the blood supply is derived from the superior muscular branch of the ophthalmic artery.

Case I.— The first patient on whom this operation was performed by me was a young man, J. F., first seen by me on June 23, 1919, at the age of 15. He had a congenital paralysis of all of the external ocular muscles of the left eye except the external rectus and superior oblique. Normal vision in right, $\frac{15}{100}$ in the left. On July 2, 1919, a partial section of the externus and advancement and resection of the left internus was done at the Jewish Hospital. A temporary improvement was secured. On July 21, 1919, when he was allowed to go home, the left eye was almost straight. I did not hear from him again until early in 1927 when he wrote to me from his home in Tupelo, Miss., that his eye was as bad as originally and that he was very anxious to have something done. On April 25, 1927, the left eye still rotated out and down almost maximal to the temporal side, at least 35 degrees. Can turn in to within 15 degrees of the median line. Movement up and down are absent. ODV = $\frac{15}{15}$. OSV = $\frac{15}{100}$. Operation of transplantation of the superior oblique tendon was suggested to him and accepted. The operation was performed at the Jewish Hospital on April 7, 1927, under general anæsthetic as follows:

The superior rectus tendon was exposed and the tendon severed about 4*mm* from its attachment, after first putting in a stay suture. A hook was then passed behind the equator and the insertion of the superior oblique easily exposed. The tendon was then dissected by first stretching between two hooks, and then completing the exposure as far as the pulley, with the closed blunt, straight strabismus scissors. The tendon was then pulled up out of the way with the strabismus hook while a sharp scalpel was slipped along the tendon from above, slid into the opening of the pulley, and the anterior portion of the cartilagenous pulley slit through completely. The tendon was then severed from its attachment to the globe, the tendon and muscle loosened backward with the strabismus scissors, about ½ inch cut off from the end, and then sutured under the insertion of the internal rectus. The tendon of the

externus was then exposed and lengthened after the method of Todd. The superior rectus tendon was then reunited and both eyes closed. On completion, the left eye was turned up, but was almost parallel otherwise.

April 10th, lid droops considerably; some edema of conjunctiva; left eye deviates up about 15 degrees. April 14th, less swelling and drooping, and not quite so much upward turning. When patient looks down and to the left, the left eye has a tendency to turn in. April 20th, still considerable redness, left eye turns up about 10 degrees and out from 15 degrees to 20 degrees. When looking down, eye rotates in about 10 degrees. Looking to the right, eye rotates in about 10 degrees. Patient permitted to go home. On May 10th, patient wrote that the condition was about the same as far as he could notice, the lid still drooping. On June 16th, he wrote that the eye was not fully opened, but was much better. This patient is to return for an advancement and reattachment upward of the new internus (superior oblique) tendon. This will give him a greater pull inward and correct the upward deviation.

Case II.— On July 10, 1927, Mrs. L. S., age 50, consulted me on account of a right third nerve paralysis. Sometime in August, 1922, the right eye began to turn out and gradually the lid drooped. She consulted an ophthalmologist, who had a general physical examination made, including a search for possible focal infection. Later she consulted another ophthalmologist and went through the same physical examination. Dr. Joseph Lyter made a complete physical examination including Wassermann, but found her in excellent condition aside from the ocular defect. She had always been myopic and in good health. ODV with -10.0 D.S. $= \frac{15}{100}$. OSV with -8.0 D.S. -0.5 D. Cyl. ax 45 $= \frac{15}{20}$. Fundus normal. Complete third nerve paralysis, right eye, except for slight function of levator when closing the left eye.

On July 28, 1927, patient was operated upon at the Jewish Hospital under gas anæsthesia. The technic was very much the same as in the first case except that a stay suture was placed at the insertion of the superior rectus for the purpose of traction, making the exposure and separation of the oblique much easier. Another change was made in the insertion. On account of the course of the muscle and tendon being above the horizontal plane of the middle of the orbit, the end of the cut tendon was stitched to a position just above the insertion of the internus, with the thought of avoiding the tendency to upward

deviation. This was justified by the result. On August 1st, there was good movement and the result looked most promising. On August 9th, the right eye could be moved as far as the median line, with no upward deviation. Slight action of the levator. On November 29th it was noticed that the right lid could be raised quite a bit if the left eye were closed, but that it drooped almost completely with both eyes open. The right eye could be moved to about 5 degrees past the center. Advised shortening of the new tendon serving as internus, and of the levator.

On December 15, 1927, the new tendon was exposed, a segment was cut off and the end was stitched farther forward close to the corneal margin. A horizontal incision was made the length of the right upper lid about the middle of the tarsus, the tarsus cleaned of orbicularis, the aponeurosis of the levator exposed for the distance of about 12*mm* above the upper border of the tarsus. Two mattress stitches were then placed through the aponeurosis and muscle above, and then deep through the surface of the tarsus about 4*mm* from its upper border, forming a tuck in the levator muscle and thus raising the lid. The excision of part of the orbicularis heightened the effect. Patient left the hospital on December 19th. On December 20th, eyes were parallel on looking straight ahead. Movement beginning inward. Lids swollen. December 22nd, lids not quite so swollen. December 23rd, stitches removed. On January 3, 1928, raises lid fairly well.

Summary.

From these two cases it would seem that we can give our patients afflicted with paralysis of the third nerve a most favorable prognosis, at least from a cosmetic standpoint. If the patient is operated upon early enough, as suggested by Jackson, there is also the possibility of restoration of binocular single vision. The experience gleaned from these two cases shows that about one inch of tendon should be excised, and that the new insertion must be placed above the attachment of the internus to avoid upward rotation, and must be sewed well forward. There appears to be very little danger of over correcting. The technical details are surprisingly simple and easy of execution.

1. Edward Jackson. "Wood's system of ophthalmic operations." Volume I., p. 741.

2. DRANSART. *Revue Général d'Ophtalmologie*, 1907, p. 229. "De la suppléance du muscle grand oblique par la muscle droit externe et par la muscle droit inférieur."
3. WHITNALL. "Anatomy of the human orbit."

ILLUSTRATING DR. MARY KNIGHT ASBURY'S ARTICLE, "A PATHOLOGIC STUDY OF TWO CASES OF OCULAR TUBERCULOSIS."

FIG. 1.

(Case 1.) Ciliary region showing site and extent of lesion. (x 10).

FIG. 2.

Area *a* in Figure 1. Destruction of ciliary body and exudate on inner surface. (x 40).

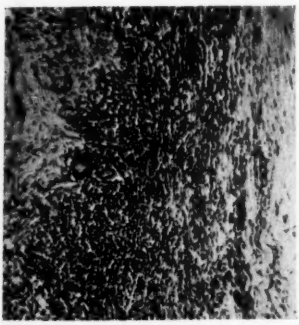

FIG. 3.

Lymphoid infiltration and giant cells on edge of conglomerate tubercle. (x 85).

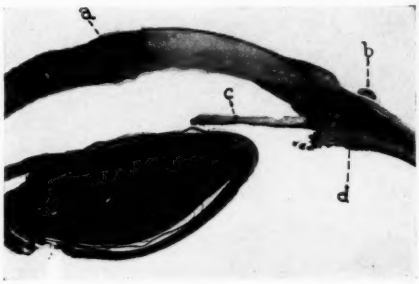

FIG. 4.

(Case 2). Anterior segment of globe. (x 10).

ILLUSTRATING DR. MARY KNIGHT ASBURY'S ARTICLE, "A PATHOLOGIC STUDY OF TWO CASES OF OCULAR TUBERCULOSIS."

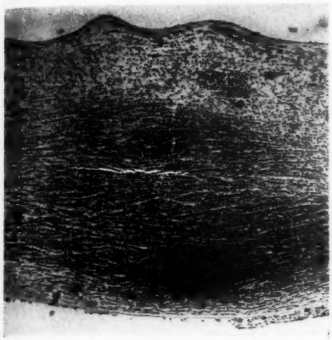

FIG. 5.

Area *a* in Figure 4. Corneal abscess. (x 40).

FIG. 6.

Area *b* in Figure 4. Tubercles at corneo-scleral juncture. (x 40).

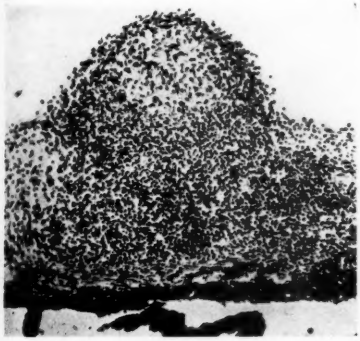

FIG. 7.

Area *c* in Figure 4. Young tubercle in iris. (x 85).

FIG. 8.

Area *d* in Figure 4. Tubercle on outer edge of ciliary body. (x 15).

A PATHOLOGIC STUDY OF TWO CASES OF OCULAR TUBERCULOSIS.

By MARY KNIGHT ASBURY, M.D.

(Section on Ophthalmology, Mayo Clinic, Rochester, Minn.)

(With eight illustrations in Text-Plates XXXV–XXXVI.)

TWO cases of proved ocular tuberculosis which have come to my attention are noteworthy because the symptoms and clinical course cannot be readily reconciled with the pathologic picture.

Report of Cases.

Case 1.—A man aged sixty-three came to The Mayo Clinic October 10, 1923, because of painful, inflamed eyes. The trouble had started a year and half previously with severe pain in the top and back of the head. A few months later, the eyes became painful and intolerant to light, and the patient was unable to work, although his general health was good. He had been treated for iritis for a year.

The condition of the right eye resembled scleritis more than active iritis. There was marked photophobia and some lacrimation. The bulbar conjunctiva was congested. Near the temporal limbus the sclera was thickened, deeply discolored and bulged slightly over an area 8*mm* in diameter. The cornea was clear, the corneal reflex normal. The iris was dark blue, was not swollen and did not contain nodules. The pupil was 4*mm* in diameter, eccentrically placed, and did not react to light because of posterior synechia. A lesion was not seen in the fundus. The ocular conjunctiva of the left eye was slightly congested. The cornea was clear except for slight vascularization along the upper margin. The anterior chamber was deep, and the iris dark blue; the pupil was slightly irregular and reacted sluggishly to light. Ophthalmoscopic examination was negative except for many

floating vitreous opacities. Examination for foci of infection disclosed only destructive arthritis of the right wrist.

Three years before this examination, he had visited the clinic because of this sore, stiff wrist. The wrist had been injured in 1918 in a fall from a horse. Two years later soreness, stiffness and enlargement of the joint developed gradually with limitation of movement. The roentgenogram showed mild destructive arthritis, presumably tuberculous. A roentgenogram of the chest showed interlobar pleurisy on the right side. The patient was given a splint for the wrist and when he returned three years later, the condition had improved greatly; there was no pain and but little stiffness.

At the second examination many plugs were expressed from the tonsils, but they were not definitely septic. The appearance of the nose suggested that partial submucous resection of the septum had been performed, but the patient said that the nose had sunk in gradually and painlessly. The Wassermann reaction was negative, the blood count was negative and the urine normal. A special examination of the pelvic organs yielded negative results. There were supernumerary teeth but no evidence of dental infection.

The patient spent four months in the hospital where a number of remedies was tried. Ten cubic centimeters of whole milk was injected intramuscularly every other day for about three weeks. At first the eye seemed to respond and the patient was more comfortable. Later the pain returned, and the margin of the cornea nearest the scleritic nodule became infiltrated. The milk injections had never produced a febrile reaction, however, and had only once caused definite leukocytosis. Typhoid and streptococcus vaccines were tried for their non-specific actions. Dionin was used to control pain but was seldom effective. Subconjunctival injections of oxycyanide and other local measures were also unsuccessful. The degree of involvement of the sclera varied considerably, and with succeeding exacerbations the infiltration of the cornea slowly progressed.

Foci of infection were repeatedly sought. Subsequent examinations of the nose revealed idiopathic destruction of the entire cartilagenous septum with the membranes intact. There was no evidence of sinus disease. Because of the destruction of the septum and destructive arthritis of the wrist, tuberculosis was believed to be the etiologic factor in the ocular lesion. During the period of treatment the vision had steadily decreased in the right eye and the patient was very anxious to have the globe removed to be relieved of the pain.

Enucleation was followed by more than the usual amount

of hemorrhage which had no untoward effect. The socket healed promptly and the patient was relieved of pain although he still had many indefinite complaints. About three months later he committed suicide.

Pathologic report.—The globe was normal in size and shape. The cornea was fairly clear except for some deep infiltration near the temporal margin. In this area, the limbus and adjacent sclera were thickened and slightly nodular (Fig. 1). The globe was sectioned horizontally along the upper and lower borders of the cornea and the central portion embedded. There was a layer of white gelatinous exudate over the region of the ora serrata in the area corresponding to the scleral thickening. In front of the equator were a few small spots of choroidal atrophy and pigment migration.

Microscopic report.—The corneal epithelium was intact but there were a few areas near the periphery where it had become thickened to fill in gaps in Bowman's membrane. The central portion of the cornea was fairly normal; the lamellæ were evenly arranged, and were not infiltrated. Near the juncture of the cornea and the sclera there was lymphocytic infiltration of the substantia propria. This was more marked on the temporal side where it involved several millimeters of corneal tissue. The infiltration had increased the width of the cornea and had destroyed or separated the lamellæ. The infiltration was patchy. There was a small amount of vascularization, more noticeable in the small areas of infiltration. On the nasal side there was only a limited amount of lymphocytic infiltration around the anterior ciliary vessels and Schlemm's canal, and in the superficial layers of the sclera.

The anterior chamber was of normal depth, the angle was open but there were lymphocytes between the fibers of the pectinate ligament. There were several nodules of lymphocytic infiltration in the iris and the stroma was diffusely infiltrated with lymphoid and plasma cells.

On the temporal side the infiltration was much more marked in the sclera than in the cornea. The sclera was thickened; the line of division between the sclera and ciliary body was lost, and the ciliary body partially destroyed; even the adjacent choroid and retina were involved. The lesion had caused most destruction in the region of the ora serrata. The outlines of the anterior part of the ciliary body were well preserved although the tissue was diffusely infiltrated; the pars orbiculus and the anterior part of the choroid were entirely destroyed, even the pigment granules of the epithelium were widely scattered (Fig. 2). There were numerous necrotic areas surrounded by walls of

epithelioid cells and separated by bands of fibrous tissue, and many foreign-body giant cells especially along the margins of the necrotic areas (Fig. 3). This area resembled a large conglomerate tubercle. Overlying this lesion the sclera was thickened. In the outer layers there was extensive lymphocytic infiltration. Many of the fibrous bundles of the sclera were destroyed and the remainder were infiltrated with lymphocytes. A layer of fibrin and lymphocytes covered the ciliary epithelium of the pars plana, became thicker over the ora serrata, replaced the epithelium and anterior part of the retina and then tapered off again. The choroid adjacent to the lesion was also thickened by the lymphocytic infiltration. Near the lesion the retina was separated from the choroid for several millimeters by a clear pink-staining exudate. The only lesion in the posterior part of the eye was slight lymphoid infiltration around some of the retinal vessels.

The lesion in this eye was situated in the pars plana of the ciliary body on the temporal side and involved the ora serrata. It was made up of small caseous foci surrounded by epithelioid cells and generously sprinkled with giant cells. Around the whole was an irregular wall of lymphocytes which infiltrated the sclera, choroid, cornea, anterior portion of the ciliary body, root of the iris and pectinate ligament. The lesion had begun as a bilateral affection clinically resembling iritis. When the patient was examined the process was active in only one eye, and resembled scleritis; the clinical course was that of scleritis and sclerosing keratitis. The pathologic examination, however, proved that it was primarily a localized lesion in the ciliary body. Although repeated attempts to stain bacilli of tuberculosis in the tissue were unsuccessful, the pathologic picture was undoubtedly that of tuberculosis.

CASE 2.—A woman aged twenty-seven was referred to The Mayo Clinic, July 13, 1925, because of pain and redness in the left eye which had first become apparent a month previously. There had been a similiar attack in the right eye nine years before, and an attack in both eyes five years previously.

The tonsils had been removed following the second attack and a microscopic diagnosis of miliary tuberculosis was made by Dr. Warthin of Ann Arbor. The case was reported at that time by Dr. Cecil M. Jack, Decatur, Illinois. The patient's general health was excellent and she had not had any more trouble with her eyes until the exacerbation which brought her to the clinic. Early in the attack a tag of tonsillar tissue was removed at her home; tubercles were

not found. The affection of the eye cleared up following this operation, however, but soon relapsed. At the time of admission the left eye had been continuously painful for four days.

The vision was $\frac{6}{15}$ in both eyes. The lids were normal. The conjunctiva of the right eye was clear. There was a dense haze in the lower nasal quadrant of the right cornea. The iris was gray-blue; the pupil was somewhat irregular and did not react to light.

The palpebral conjunctiva of the left eye was faintly injected and there was circumcorneal injection around the lower half of the limbus. The pupil was partially dilated and very irregular, due to posterior synechia. In both eyes the media were clear except for pigment deposits on the anterior lens capsule, and the eye-grounds were normal. Chronic recurring iridocyclitis was diagnosed. Examination did not reveal any outstanding focus of infection. In childhood drainage or removal of cervical glands had been performed. Healing was complete but the scar was visible. Heart and lungs were normal. A roentgenogram of the chest was negative. Clinical evidence of tuberculosis could not be found. Intracutaneous tuberculin test (Mantoux) gave no reaction. Urinalysis and blood Wassermann reaction were negative. The tonsils were cleanly out, the nose was clear but the roentgenogram showed a cloudy right antrum and slight clouding of the frontal sinuses. Investigation of the sinuses did not reveal infection, however, and the nose was considered negative as a focus. Although the history of pelvic infection was negative the cervix was investigated as a possible focus. The patient had been married four years and had had one pregnancy with normal delivery. The child was eighteen months old. Smears from the cervix showed many pus cells, and many gram-positive cocci. Culture showed streptococci, gram-negative bacilli and large gram-positive bacilli.

The first attack in 1916 cleared up after six months of tuberculin therapy. In the second attack the patient had improved slightly on administration of tuberculin, then had grown so much worse that it was discontinued. This time improvement followed the removal of tonsillar tissue from which a microscopic diagnosis of miliary tuberculosis was made. Since the examination at the clinic in 1925 did not show evidence of tuberculosis, even the intradermal test being negative, and the tuberculin had been used unsuccessfully in a previous attack, it was decided to try a vaccine made from the cultures from the cervix.

The patient returned home and the vaccine was sent to her physician for administration. One course was given.

The eye cleared up entirely and remained so for nearly two years. During that period she had another child, who was six months old when the patient returned to the clinic in August, 1927. For two months the right eye had been red and very painful. Six intramuscular injections of milk had given good febrile reactions but had not relieved the pain. Atropine had been used constantly. The eye had gradually improved for several weeks, but a severe exacerbation of pain had precipitated the patient's return to the clinic.

The palpebral conjunctiva was clear and there was no definite circumcorneal injection. The cornea was slightly hazy, the iris was discolored, the pupil irregular, and the tension decreased to palpation. The left eye was practically normal in appearance. The pupil was round, regular and reacted promptly. A diagnosis was made of recurrence of iridocyclitis of the right eye.

The patient's general health was good although she complained of lack of endurance. A considerable amount of leukorrhea had persisted for several months after childbirth. At examination the margin of the external os was slightly reddened; there was no erosion. The uterus and adnexa were normal. Streptococci were recovered from the cervical cultures. Although the evidences of infection were slight in view of the seriousness of the condition of the eye, a pelvic operation was thought to be justified. Following the operation the eye gradually improved, the pupil dilated, pain disappeared, and vision was clearer. The administration of a vaccine made from the streptococci cultured from the cervix was begun and continued by her home physician. When the patient was dismissed two weeks after operation, the eye was in fairly good condition and there was no pain. The sclera on the temporal side of the cornea was slightly infiltrated and there were three or four small gray nodules in the adjacent cornea.

The patient returned six weeks later with severe inflammation of the right eye of three days' duration. After she left the clinic the right eye had continued to improve until it was practically free from inflammation and vision had increased so that she could read large print with the affected eye. She had received increasing doses of vaccine weekly, having had seven in all. Two days after the last one the eye became red and on the following day very painful. She had not had symptoms referable to the pelvis and on the whole felt better than usual.

Vision was reduced to light perception in the right eye, left $\frac{4}{4}$. In the center of the right cornea was a yellow spot *2mm* in diameter, surrounded by a cloudy zone fading

toward the limbus. The iris was very muddy; the outlines of the pupil were slightly obscured, but appeared to be irregular and the pupil did not react. Nodules were not present in the sclera. The tension was reduced. Corneal abscess was diagnosed. The eye was enucleated the following day and the socket healed promptly. Two months later the patient reported that she was feeling extremely well.

Pathologic report.—The globe was normal in size and shape; the corneal surface was smooth, the corneal substance diffusely hazy. In the center was an opaque yellow area *2mm* in diameter. There were a few deep, straight vessels in the cornea arising at the lower limbus. The iris was very indistinct. It appeared to be grayish, retracted and bound down along the pupillary border. The sclera adjacent to the temporal limbus was purplish. The remainder of the globe appeared normal (Fig. 4).

Microscopic report.—The cornea was thicker than normal, especially the central portion, due to cellular infiltration. The epithelium near the periphery looked healthy, but it became thinner toward the center and disappeared for a short distance. In the thinned portion the deepest layer was vacuolated and a few leukocytes lay between the epithelium and Bowman's membrane, which was intact. Practically the whole of the substantia propria was infiltrated with polymorphonuclear leukocytes. The infiltration was densest in the center of the cornea beneath the break in the epithelium, and the lamellæ here were opaque and stained diffusely with hematoxylin (Fig. 5). The leukocytic infiltration practically disappeared toward the limbus where, on one side, there was widespread vascularization with accompanying perivascular lymphoid infiltration. On the other side the polymorphonuclear cells were replaced near the limbus by a dense infiltration of lymphoid and epithelioid cells, some arranged in typical tubercles (Fig. 6). In some sections this type of infiltration extended to the center of the cornea in the deep layers of the substantia propria. Typical tubercles even containing giant cells were seen. Descemet's membrane was pushed back and thrown into waves by this infiltration, but remained intact.

There were adhesions of the periphery of the iris to the cornea. In places the adhesions and the base of the iris were densely infiltrated with lymphoid and epithelioid cells. The iris was rather thick due to diffuse infiltration of the stroma with lymphoid and plasma cells. There were also discrete nodules, some of lymphocytes only, others containing a nucleus of epithelioid cells. One projected far beyond the surface of the iris into the anterior chamber (Fig. 7). In some places the pupillary margin was fastened

to the lens capsule and there were also evidences of old broken posterior synechia. The lens was normal.

In the connective-tissue stroma of the ciliary body were numerous lymphoid and plasma cells and also a few circumscribed areas of dense lymphoid infiltration. One area adjacent to the sclera contained some endothelioid cells and a large giant cell (Fig. 8). The most posterior of these tubercles occured at the ora serrata. There were no lesions in the posterior part of the eye.

At the juncture of the cornea and the sclera and in the ciliary zone, particularly on one side, the sclera was thickened and largely replaced by tubercles, collections of epithelioid cells surrounded by lymphocytes. Giant cells were seen, especially in the episcleral nodules. The posterior part of the sclera was normal. All attempts to stain bacilli of tuberculosis in the tissue failed. The scleral tubercles were the most typical and apparently the oldest seen in the eye. Those in the iris were young. The whole process, however, looked fairly recent, not more than a few months old. In spite of the history of other attacks years before, no definitely old or scarred tubercles were found. There were a few small areas between the active scleral tubercle where the tissue was more cellular and the collagen fibers less dense and not as evenly arranged as in normal sclera, but these changes were slight and were hard to evaluate when seen in such close proximity to active lesions.

Comment.

The clinical picture was that of recurring chronic iridocyclitis. The signs of scleral involvement occurred late and never were prominent. The microscopic picture was typical of tuberculosis involving the anterior part of the sclera and secondarily extending into the cornea, iris and ciliary body. The pathologic changes were similar to those described by Chou, in a case studied by him which also was clinically diagnosed chronic iridocyclitis. The involvement in his case was more extensive than in the one reported here, but the distribution was the same and was similarly interpreted as a primary infection of the sclera probably blood-borne, with secondary involvement of the adjacent structures. The corneal abscess in my case was an incidental lesion dependent on the lowered vitality or nutrition of the eye, and not characteristic of tuberculosis.

Verhoeff advanced a theory, which he believed to be

supported by clinical observation and experimental work, that the infection of the sclera is by way of the filtration angle, and that the bacilli are excreted through the intact ciliary epithelium along with the aqueous humor, and are carried by its current into the filtration angle. From the angle they have access to the sclera, the cornea, and the ciliary body through the spaces of Fontana which are continuous with the perivascular lymph spaces of the anterior perforating vessels, with the lymph spaces of the cornea, and with the suprachoroidal space. In support of this view he described minute exudates of epithelioid cells on and in the ciliary epithelium. I was not able to find any of these exudates in my cases. The infection in the iris may have been carried by the aqueous humor, but only late after the scleral process was well advanced, for the tubercles in the iris were recent and scars of old ones were not found.

In conclusion I repeat one of the points made by Chou, that tuberculous deep scleritis may exist without characteristic signs of scleritis, and add that tuberculous cyclitis may simulate scleritis.

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SOME MODERN PREPARATIONS USED IN THE TREATMENT OF GLAUCOMA.

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1. Adrenalin and Glaukosan.

WHILE the subject of adrenalin therapy is generally associated with the name of Hamburger, since he has done more than any other man to focus attention on its advantages, it should be remembered that a good deal of work had been done on the subject by others before his first published report in 1923. Darier and Landolt had reported some results in glaucoma by instillations of adrenalin in 1899, and in 1900 to 1904, Wessely reported on extensive animal experiments with adrenalin. He found that with the ordinary 1:1000 solution instillation was ineffective, but that subconjunctival injections of the same solution produced maximal mydriasis accompanied by a fall in tension. When a 1% solution was used, the same effect could be produced by instillation. He did not try the method on the human eye, but in 1909 Rubert produced a marked fall of tension in a normal human eye, lasting six to seven days. In 1914, Erdmann for the first time reported the use of subconjunctival injections of adrenalin in glaucoma, and recommended its use in that condition, in conjunction with miotics. Kollner, in 1918, confirmed his results. Knapp, in 1921, investigated the reaction of sixty-five glaucoma cases to repeated instillations of adrenalin. All but five showed mydriasis and twenty showed a decrease of tension. This difference in the reaction of glaucomatous eyes from that of normal eyes seemed to Knapp to indicate an

increased sensitivity to sympathetic stimuli. In the same year Fromaget noted the immediate lowering effect on the tension of retrobulbar injections of novocain-adrenalin before glaucoma operations, which made operation unnecessary in some cases. Aubaret used the same method in a number of cases. Since 1923, besides some thirteen communications from Hamburger himself, numerous reports have appeared, chiefly in the German journals, on the subject of adrenalin therapy, only a few of which it will be necessary to mention. Gradle in 1924, referring to Erdmann's work and to some earlier work of his own, was the first American to report on the use of adrenalin injections. He gave his results in sixteen cases, and recommended the method especially in glaucomatous iritis and after filtering operations which had not given sufficient effect. In America besides Gradle's series, Pischel has reported on eighteen, King on three, and MacNie, Watson and others on one or more cases in which adrenalin proved of value. The reviewer, in 1928, reported on some cases of acute glaucoma produced by adrenalin. Hamburger at first used subconjunctival injections of three to five minims of 1:1000 adrenalin. On account of the sudden increase in blood pressure observed in a few susceptible patients with this dosage, he next recommended the same injection of dextro-rotatory adrenalin, to which was added an optically inactive substance produced in the synthetic manufacture of adrenalin. This solution, which he called Glaukosan, was devoid of unpleasant systemic effects, but, like ordinary adrenalin, was effective only on injection. He then had prepared a 2% solution of the usual levo-rotatory adrenalin, to which the same optically inert substance was added, and this solution, which he called Linksglaukosan or Glaukosantropfen, was effective by instillation. In using it, the patient is placed in the lying position, the lids are held open, and two drops are placed in the inner angle, after which the lids are held open for thiry seconds. This is repeated three to five times at fifteen minute intervals, according to the effect desired. This produces a marked blanching of the conjunctiva extending to the skin of the lids, with maximal mydriasis and a fall in tension. Being twenty times the strength of the ordinary adrenalin solution, this preparation must be reserved for

instillations only, and never injected, for fear of systemic effects, but no such effects are observed after its instillation. Thiel, besides subconjunctival injections of adrenalin, used a salve made with vaseline and adrenalin 1:1000, which patients could use daily, if necessary. Gradle, after having used subconjunctival injections, in 1925 advised the simple method of soaking a cotton pledge with four minims of adrenalin, and leaving this in the upper cul-de-sac for three minutes. Enough absorption occurred when it was used in this way so that the same effect on tension was produced as by injection, with absence of systemic effects, and simplification of the preceding anesthesia to the instillation of two drops of 2% butyn. (The reviewer can vouch for the simplicity of this method, and has found it as effective as injections, after using it some one hundred times.) About the same effects have been reported from the use of these different methods. Gredstedt in a recent comparison of adrenalin injections, and Glaukosantropfen, found that the decrease of tension from adrenalin in simple glaucoma was more marked and more lasting than that of Glaukosantropfen.

The effects are, first mydriasis, which begins a few minutes after the first application, reaches a maximum in thirty minutes, and lasts six to twelve hours. This mydriasis is strong enough to break up fresh synechiæ which have resisted atropine and hence is of value in some cases of iritis without increased tension. In some cases, the tension at first remains unaltered, or even rises a few millimeters. In others, the tension begins to fall almost at once, but in most a significant fall is not noted for one to three hours, and the greatest reduction is seen the following day. The amount of reduction varies, some cases showing none, while in others a tension of 9 to 10*mm* (Schiotz) is reached. Of a series of sixty-one glaucomatous eyes carefully observed by Vannas, thirty-six showed a significant fall of tension which reached normal or below in twenty-five of these. In one eye, a fall of 42*mm* was recorded but the average reduction was 18*mm*. Where the initial tension was not over forty, all the eyes in his series were reduced to normal tension, and some eyes with higher tension were also normalized, among them one showing an initial tension of sixty.

The duration of the reduction in tension also varies greatly. In Vannas' series, of those in which normal tension was reached, it lasted one day in a third of these, two to three days in a third, and over three days in the remaining third, lasting as long as six to twelve days in six of these eyes. In a series of forty eyes reported by Rentz, the usual duration of normal tension was one to three days, but was four days in three eyes, five days in one, and eight days in one. Thiel found it necessary to repeat the treatments frequently, so used a 1:1000 ointment, and by having the patients themselves use this several times a day, was able to keep the tension normal for several weeks, but this method of treatment is open to an objection which will be discussed later. On account of the great individual differences in the reactions to the treatment, it is impractical to control most cases of glaucoma by adrenalin alone. One great advantage of the treatment, however, is that, when combined with the use of miotics, the tension may be kept normal where miotics alone have proved incapable of such a result. Hamburger recommends using glaukosan eight to ten days, then returning to miotics, and when these become ineffective, using glaukosan again. He states that he has been able to keep the tension normal in many eyes for years by this method. Rentz found that after an adrenalin injection miotics were much more effective than before, and in six eyes was able to keep the tension normal for five months by these means.

As to the types of glaucoma in which adrenalin is indicated or contra-indicated, most authors agree that it is ineffective in absolute glaucoma, and in inflammatory and hemorrhagic glaucoma. Hamburger, Rentz, Gradle, and Thiel recommend it as of especial value in glaucomatous iritis, where fresh synechiæ are broken up by the mydriasis at the same time that tension is reduced. Wegner, however, having used it in six such cases, saw good results in only two, and harmful effects in two. Pischel found the results uncertain and the treatments painful in this type of glaucoma. Jess, Seefelder, Marchesani, and Hildesheimer saw acute attacks of glaucoma follow its use in such cases, and Vannas, out of five cases, saw no effect in two and a marked rise of tension in the other three. He believes adrenalin is contra-indicated in any

inflamed eye. Its greatest field of usefulness is in simple glaucoma, where it will serve, in a large percentage of cases, to reduce the tension before operation, or, when used with miotics, may allow postponement of operation for long periods, and in some cases obviate it altogether. It is especially valuable in cases with a tension which remains from 30 to 35*mm* Schiotz, in spite of miotics, in which an occasional adrenalin treatment will bring it down to normal, and allow miotics to keep it down for variable, but sometimes considerable lengths of time. When confronted by an elderly patient who has lost one eye following a previous operation and who has a small field in the remaining eye, the surgeon may be most grateful for such an addition to his resources.

The experience of the reviewer and his associates covers fifty treatments, the records of which are complete enough to be of any value. In about two-thirds of these, packs were used, in the other third subconjunctival injections. In fifteen instances the treatment failed to produce a decrease of tension sufficient to be of value. These failures were in cases of iritis glaucomatosa, two, inflammatory glaucoma, two, absolute, one, secondary, one, acute, one, and simple, nine. Among these occurred four attacks of acute glaucoma, two being in simple, one in secondary, and one in inflammatory glaucoma. In all the other cases, most of which were of simple glaucoma, a marked fall of tension occurred, the average decrease for the whole series being 10*mm*, while individual falls of 20, 23, and 24*mm* were recorded. In all but three of these, the tension reached normal. The tension was not recorded often enough to determine the average duration of decreased tension, but in thirteen it remained normal one week or more and in five one month or more, miotics being used also during this time. Many left observation with tension still normal, and several are still under observation. This experience agrees with that of other authors who report best results in simple glaucoma, and very few useful results in glaucoma of other forms.

The occasional systemic effects from adrenalin injections, while alarming, have not produced any permanent ill effects, and may be avoided altogether by the use of adrenalin packs or Glaukosantropfen. The only other complication which

may occur is the provocation of an acute attack of glaucoma. As has been seen, this is especially liable to occur in secondary glaucoma or in any case with an inflamed eye. While it occurs rarely in simple glaucoma, it does occur. The reviewer has seen four such attacks and Rentz, Stock, Hamburger, Hegner, Lohlein, Elschnig, and Vannas have also reported its occurrence in simple glaucoma. Stock saw it in three cases out of twelve in which adrenalin was tried and condemned the method on that account. While these attacks, so far as their reports show, have all been controlled without permanent ill effects, they are painful and alarming both to patient and surgeon. They occur in the first few hours after the treatment, during the period of mydriasis and are apparently the result of the mydriasis. The mydriasis may be minimized or prevented altogether by the free use of miotics before treatment and for several hours afterwards, without affecting the desired fall in tension. Ring advises miotics after adrenalin treatments to guard against acute rise of tension. Hamburger has suggested using eserine in this way, and has recently offered a new preparation, called amin-glaukosan, which controlled one such attack provoked by adrenalin. Two drops of eserine at five minute intervals before treatment, one drop every twenty minutes for the first hour afterwards, and one every half hour for the next two hours is usually effective in minimizing mydriasis. The patient should be watched for two hours after treatment and instructed to use enough eserine after this time to keep the pupil small. This is also usually effective in controlling the pain sometimes complained of, which may indicate a slight temporary rise of tension.

The modus operandi of adrenalin treatments is under discussion. At first thought to result from the pressing of blood out of the eye by the vaso-constriction produced, it would now seem that this explanation is insufficient, since the greatest fall of tension occurs when the vaso-constriction has given place to a secondary vaso-dilatation of the uveal vessels. This hyperemia is thought by Hamburger to be the beneficial factor, by promoting proper tissue functions and replacing venous stasis by arterial hyperemia. Vannas is content with believing that the vaso-constriction sets up some unknown changes in the tissues which result in decreased tension.

The suggestion of Knapp that the mydriasis is the important factor, allowing fluid collected in the posterior chamber to pour out into the anterior chamber, would seem to be inadequate, since the greatest fall of tension occurs the following day, long after the mydriasis has disappeared, and also because the fall of tension occurs where mydriasis is prevented by miotics.

An interesting fact is that adrenalin injections produce in the opposite untreated eye a slighter but definite reduction of tension. While this is not of much therapeutic importance, it arouses interesting speculations as to the nervous control of the tension of each eye being somewhat dependent on that of the other. The amount of adrenalin absorbed into the circulation would seem to be insufficient to produce such an effect, though some have explained the phenomena in this way.

2. Amin-glaukosan.

In acute glaucoma, as has been seen, adrenalin treatments have proven of little value, although a few authors, as Macnie, Ring, Pischel, and Gradle, have reported isolated cases. Hamburger, Vannas and others consider adrenalin contra-indicated, on account of the increased mydriasis, which makes operation more difficult, and may further increase the tension. In this condition, histamin, or amin-glaukosan, may prove itself of some value, although too few observers have as yet reported upon its use to justify very definite claims for it. It is a protein split product derived from histidin, a principal source of which is ergot. It was first described by Dale in 1910, and was used, like ergot, in obstetrics. It exercises a paralytic effect on the sympathetic nerve-endings, is very toxic when injected, and when instilled in the concentration recommended by Hamburger, exercises an effect on the pupil which, according to him, qualifies it as "the strongest miotic." The previously used 1:1000 solution proving ineffective, except by injection, which was very painful, Hamburger prepared a 10% solution which he called Amin-glaukosan. One drop of this instilled in the conjunctival sac, the lids being held open for thirty seconds afterwards, produces a miosis sufficient to contract the pupil dilated by atropine to pin-point size in seven to fifteen minutes. It produces chemosis of the con-

junctiva, hyperemia of the uveal tract, and marked flushing of the whole face lasting three to six hours. Two drops of holocain should be used before instillation and the patient should be in the lying position. Using this preparation, he reports one case of acute glaucoma with tension of seventy, in which three drops at intervals of several hours, brought the tension down to twenty-four after twenty-four hours, where it was kept by miotics for four months. A second case is evidently the same one mentioned above in which amin-glaukosan was successful in aborting an acute attack brought on by glaukosan. He reports another case of secondary glaucoma, in which Fehr, using his original preparation of 1 to 1000 by subconjunctival injections, aborted the attack. He has recently advised its use after glaukosan drops, to avoid the possibility of an acute rise of tension, and was successful in avoiding this in two patients in whom, without amin-glaukosan, it had occurred.

Jäger reports one case of acute glaucoma in which the use of amin-glaukosan had no effect on the pupil or tension, and produced so much chemosis and swelling of the lids that operation had to be postponed forty-eight hours. It will be seen that not enough experiences with Histamin have been reported to justify any definite claims for it in acute glaucoma. Definite drawbacks to it are the intense congestion and chemosis always produced, and the pain usually complained of.

3. Hypertonic Solutions.

Another method of reducing tension in acute glaucoma is the use of hypertonic salt solutions, systemically. This method, which was first used by Cantonnet in 1904, was suggested as a means of making the blood hypertonic, with the idea that this condition would cause absorption of fluids, including the intraocular fluids, into the blood stream, and would so lower the tension. Hertel, in 1913, reported on three cases in which the intra-venous injection of 150 to 200*cc* of 10% sodium chloride reduced the tension, and those who have used the method since have usually referred to the technique of Hertel. Pletnewa reported on twenty cases so treated, with decrease of tension in all. Three cases were given the same amounts of salt solution by mouth, but in

only one of these did a fall of tension result. The effect on tension is naturally only a temporary one, so the method is chiefly of use in preparing patients with high tension for operation, or for controlling tension where operation must for some reason be delayed. Weekers reported good results in seven cases of acute glaucoma, the tension being reduced so that operation could be delayed several days in some cases. He saw no effect on tension in four cases of simple and two of secondary glaucoma. Weekers used only 5*cc* of 30% salt solution, which is not a sufficient amount to have much effect on the osmotic pressure of the blood. Göslich, using Hertel's technique, obtained reduction of tension in most of his series (19 cases), the amount of reduction varying from 7 to 21*mm*. Operation was done one to one and half hours later. One case of hemorrhagic glaucoma, one of acute glaucoma, and several of other types, were refractory. Göslich removed 150 to 200*cc* of blood before his injections, and in patients where this was not considered safe, obtained the same effect from the same amount of salt solutions given by mouth.

Duke-Elder gave 35 to 50*cc* of 30% salt solution, and reports good results in four cases. In one case of acute glaucoma the tension was brought from 95 to 38*mm* in six hours by this method, when operation was performed. In a case of secondary glaucoma the tension decreased from 58 to 20 and remained about 30*mm* for several months. He advises more concentrated solution, which does not require a large enough amount of injection-fluid to increase the blood volume appreciably, and so produces more rapid hypertonicity. Aside from a fall of blood pressure which has caused no serious results, and marked discomfort from the thirst for several hours, no inconvenient effects of the injections have been noted. The injection should be given slowly, requiring ten minutes for the 30% and thirty to forty-five minutes for the 10% solutions. Water should not be given afterwards. Duke-Elder summarizes the indications for the method as follows:

1. In acute glaucoma, in conjunction with miotics, before operation.

2. To reduce high tension before any operation. Besides lessening the danger of vitreous loss and hemorrhage, reduc-

tion of tension allows the absorption of local anesthetics, which are ineffective in eyes with high tension.

3. In iritis glaucomatosa, before using atropine.

4. To facilitate ophthalmoscopic examination where the cornea is steamy from increased tension.

5. Is of possible value in any intra-ocular inflammation by withdrawing fluids from the eye, these being replaced by fresh fluids from the blood stream, bearing increased anti-bodies or drugs which have been given. It would act theoretically like a paracentesis.

As no traumatism is inflicted on the eye itself there is no danger in using this method in any form of glaucoma, and it does not increase the technical difficulties of operation, as amin-glaukosan may do.

Solutions of 30% glucose have been given by Sansum to produce the same effect on the blood but large amounts must be given (300 to 500*cc*). Magnesium sulphate in large doses by mouth may also extract enough water from the blood by way of the intestinal tract to lower the intra-ocular tension and is used by neurological surgeons for the same effect on intra-cranial tension. The effect of giving these solutions by mouth, while less rapid than when the intra-venous route is used, is more prolonged. Duke-Elder suggests (p. 24) that the effect of intra-venous injections is enhanced and prolonged by the addition of some colloid such as gum arabic to the injection fluid.

Some of the newer diuretics, such as Novazurol and Salyrgan have also been successful according to Hamburger, in reducing tension.

4. Tyramin.

Besides histamin or amin-glaukosan, another derivative of ergot, tyramin is being tried in glaucoma. This has in many ways the opposite effect of histamin, being a vaso-constrictor and sympathetic stimulant, and so dilating the pupil. While, according to vom Hofe, tyramin alone has little effect on the tension, when combined with histamin in the proportion of fifty parts tyramin to one part histamin, it produces maximal mydriasis with a fall of tension greater than that produced by adrenalin. The fall of tension is also marked in the opposite

eye, and lasts longer than in the injected eye. $\frac{1}{10}$ to $\frac{5}{10}cc$ are injected sub-conjunctivally. This combination of tyramin and histamin has been prepared by Bayer under the name of "Tenosin." Thiel saw a reduction of tension in several cases produced by "Tenosin," but found that in the same case, ergotamine, a drug to be described later, produced a more marked effect, so gave up the use of "Tenosin."

5. Calcium.

Based on altogether different principles is the treatment of glaucoma by calcium. Chiari and Januschka found that calcium decreased the permeability of the capillaries, so preventing the edema produced by irritating agents such as abrin. Weekers in 1912 on these theoretical grounds, tried giving calcium chloride internally (3 grams daily) and found that some cases of glaucoma which had resisted miotics could be controlled in this way. The effects were not constant, but valuable in some cases. Gowland in 1917 reported relief of pain in five cases of acute glaucoma by the injection of 2*cc* of 5% calcium chloride intra-muscularly. Alt in 1919 reported in 12 cases of all types of glaucoma, with favorable results in most of these. He gave calcium chloride by mouth. Kleiber tried the local effect of calcium on rabbits' eyes. Subconjunctival injection of $\frac{1}{2}cc$ of 3% afenil or 1*cc* of 2% $CaCl_2$ produced a marked fall of tension lasting in the latter case for a week. That this was due to decreased permeability of the ciliary epithelium or the capillaries seemed probable since such injections markedly delayed the appearance in the aqueous of fluorescein given intravenously. Subconjunctival injections produced severe inflammation but instillations of 10% $CaCl_2$ produced as marked a fall of tension without signs of inflammation. No experience with this use of calcium in human glaucoma is reported. Vom Hofe reports that the use of calcium chloride and adrenalin together produces a fall of tension much more marked than that of adrenalin alone, lasting as long as ten days.

6. Barium.

Another inorganic salt whose effect on the (glaucomatous) eye has recently been studied is Barium chloride. This is

known to produce marked contraction of smooth muscles and vom Hofe in 1926 found that sub-conjunctival injections of .2*cc* of a 2% solution produced maximal miosis in rabbits even after atropine. Barium and Adrenalin together have much more effect than adrenalin alone while barium alone has no appreciable effect on tension. Subconjunctival injections of barium produced marked chemosis and falling of the hair around the eye. No reports of the use of barium in human glaucoma are at hand.

7. Ergotamine (Gynergen).

A third derivative of ergot which has been used in glaucoma, and the use of which has received more justification from clinical experience than either histamin or tyramin, is ergotamine. This contains the active principle of ergot most desired in obstetrics, and under the name of gynergen, is used subcutaneously in a dosage of $\frac{1}{100}$ grain or by mouth $\frac{1}{60}$ to $\frac{1}{30}$ grain. Besides causing contraction of the uterus, it raises the blood pressure. It depresses the sympathetic nerve-endings and its use in glaucoma is based on the idea that hypertension is due to hyperactivity of the sympathetic system, and that such a drug would decrease the permeability of the vessels. Thiel and Römer and Krebs found that sub-conjunctival injections of 1% solution produced slight miosis and fall of tension in the rabbit's eye.

Thiel reports on ninety cases of glaucoma in which ergotamine was used by subcutaneous injection of .00025*gm* or oral administration of one to 3 tablets of .001*gm*, doses by each method being repeated three times a day. Rest for one hour after treatment is advised to avoid systemic symptoms observed in a few cases. A summary of results in these ninety cases is not given but a few illustrative cases show that in simple glaucoma the tension could be kept normal by oral administration of ergotamine for several months, and that after the first three to four weeks miotics which had been ineffective, were so much more effective that the ergotamine could be discontinued. In one of these cases the tension sank from 70 to 24 after one subcutaneous injection. In iritis glaucomatosa and some cases of secondary glaucoma where iritis was present, good results were obtained, and in one of

these an acute attack of glaucoma was aborted by one injection. No effect was secured in absolute glaucoma. In inflammatory glaucoma a fall from 72 to 24 was observed in one case, but after remaining near normal for three days, tension rose to its former level in spite of the continued use of the drug. In this condition, while failures may be expected, ergotamine may at least be tried without danger of producing an acute attack of glaucoma such as may occur after adrenalin. Römer and Krebs observed reductions of tension in glaucoma produced by ergotamine, but not so great as would be produced by adrenalin. Heim confirms Thiel's observations in a series of cases and also found that after using fairly large doses for a variable time, much smaller amounts were effective in keeping the tension normal, when combined with the use of miotics. The advantages of any method of treatment that can be given systemically, without traumatism to the eye, are obvious in certain cases of glaucoma. In sympathetic ophthalmia, for instance, when the pupil is bound down and rise of tension occurs, miotics are contraindicated, and operative interference is dangerous. In two such cases, the reviewer has seen at least temporary effect from ergotamine. In one case of glaucoma secondary to a cataract operation and another complicating a severe iridocyclitis the effect of ergotamine has been of great value. Poos has recently offered evidence that ergotamine is not a sympathetic depressant, but a stimulant of both the sympathetic and para-sympathetic systems. Hess shares this view since the drug is effective when the sympathetic is paralyzed.

8. Pituitrin.

Besides adrenalin, another glandular extract, pituitrin, has been used in the treatment of glaucoma. Müller in 1924 and Samojloff in 1927, on the basis of Krogh's proof that pituitrin increased the tone of the vessels without the secondary vasodilatation produced by adrenalin, investigated its effect on the intra-ocular tension. Müller found that dropping pituitrin in the normal eye produced a deeping of the anterior chamber, which he interpreted as due to an increase in tone of the dilator iridis, although the pupil remained small. In three cases of glaucoma, a fall of tension was produced by instilla-

tions five times a day. Samojloff found that subconjunctival injections of .3 to .5*cc* of pituitrin (Parke-Davis) produced a marked fall of tension without the preliminary rise of tension seen with adrenalin, and without dilatation of the pupil. In rabbits, the fall amounted to 10 to 14*mm*. Injections in glaucoma patients after holocain instillation, were painless, but caused slight chemosis. A rise of blood pressure was observed, which had disappeared after twenty minutes. The decrease of tension was most marked on the second or third day after injection, and remained low for several days without the use of miotics. In one case of simple glaucoma tension decreased from thirty-five to twenty, and remained at twenty-five for eight days, when pilocarpin, previously ineffective, kept it at twenty-two during further observation. In a case of acute glaucoma with tension of sixty, this fell to twenty-two after one injection of .5*cc* and after a rise to forty on the fourth day, was kept between twenty-five and thirty for two weeks, when operation was performed. In three cases of advanced decompensated glaucoma pituitrin produced decrease of tension, but not enough to avoid operation.

Summary.

While the meagerness of clinical reports concerning many of these newer methods of treatment prevents any definite judgement on their value in glaucoma, some of them present interesting possibilities well worth further investigation. As to the danger of attempting their use, the principal ones that must be borne in mind are that of provoking acute glaucoma by adrenalin or glaukosan treatment, especially in eyes that are inflamed, and that of troublesome inflammation and chemosis which may be caused by amin-glaukosan or histamin. Enough evidence has accumulated to decide that adrenalin is of considerable value in simple glaucoma, in conjunction with miotics, in delaying operation, and that in some cases its use will obviate the necessity of operation, which is especially desirable where the field is already small. It may also be said that any of the methods of making the blood hypertonic, especially intravenous injections of 30% salt solution, have proven of definite service in reducing the tension until operation can be performed under favorable circumstances. The

internal use of calcium and especially the internal or subcutaneous use of ergotamine would seem to present no dangers, and a trial of them in conditions where operation is undesirable seems indicated. The results of further experience in clinics where experimental therapeutics can be carried out with histamin, tyramin, barium and pituitrin, should be watched for with interest. While the mode of action of most of these drugs is still a matter of great uncertainty, investigations of them now under way in several clinics may some day throw light on the nature of glaucoma itself.

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OCULAR SYMPTOMATOLOGY IN DENGUE.[1]

Based on an Analysis of 1241 Cases.

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DENGUE fever is an acute, insect-borne disease of unknown etiology which is endemic in the tropics, but which at times becomes epidemic in character. In the hotter portions of the year it may spread to temperate regions and assume an epidemic form. It is a mosquito borne disease, and available experimental and epidemiological evidence indicates that it is transmitted from person to person only by the adult female of ædes (stegomyia) ægypti.

The typical case is characterized by sudden onset; an initial erythematous rash; severe pains in the head and about the eyes, pains in the trunk and limbs; a fever of short duration, showing a fairly typical curve, and usually a well-marked leucopenia. A terminal rash appears in most cases. The acute stage of the disease covers an average period of six days. It is important because of the large numbers affected in epidemics and the prolonged period of convalescence which follows it. It is practically without mortality. In its pandemic tendencies it resembles influenza.

It must be differentiated from yellow fever, trench fever, measles, scarlet fever, influenza, Brills disease, papatacci fever, Rocky mountain spotted fever, meningitis, malaria,

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the early stages of smallpox, and in addition, from certain dengue-like fevers which are common to the tropics.

All attempts to isolate a causative organism in dengue have given more or less discouraging results; but, inasmuch as it is an insect borne disease, it is natural to infer that the causative organism is present in the blood stream. Ashburn and Craig (1) have proved that the unidentified causative agent is present in the blood stream by producing dengue fever in non-immune volunteers through the injection of blood from patients with the disease. Cleland, Bradley, and MacDonald (2 and 3) have verified the experiment of Ashburn and Craig on the blood transmissibility of the disease, and have also obtained experimental evidence that the infectious agent is present in the blood stream of dengue patients as late as the eighth day of the disease. Siler, Hall, and Hitchens (4) have recently verified the insect transmissibility of dengue by the production of the disease in volunteers through the agency of ædes (stegomyia) ægypti mosquitoes that had bitten dengue fever patients.

It is generally conceded that the ocular symptomatology, which forms such a striking feature of the early stages of dengue fever, is important in the differential diagnosis of the condition, for, not infrequently, certain of the ocular symptoms appear in advance of any other definite diagnostic signs of the disease, and, in the presence of an epidemic, are strongly suggestive of the onset of dengue.

During the summer of 1923, an epidemic of dengue fever occurred in the vicinity of San Antonio, Texas, affecting the military as well as the civilian population of the city. During the epidemic, twelve hundred and forty-one patients with dengue fever were admitted to the Station Hospital at Fort Sam Houston, where, at that time, the writer was in charge of the Eye Clinic. An epidemic of dengue fever in the preceding year had focused attention on the ocular symptomatology associated with the disease and caused us to make a special study of the eye symptoms in this new group of patients. These investigations disclosed certain symptoms referrable to the eye which were striking in character, surprisingly constant in occurrence, and of sufficient importance to warrant the detailed description which will follow.

The first symptom of note was retrobulbar pain. This symptom was present in every patient early in the disease, was usually quite intense, and formed one of the predominant characteristics of the illness. It was almost invariably described by the patient as being behind the eyes. Riley (5) suggests that the post-orbital pain, headache, and other phenomena such as loss or perversion of taste, vertigo and mental depression, which characterize the majority of cases of dengue, are of central origin. Siler, Hitchens, and Hall (6) agree that an analysis of experimental cases, as well as those occurring spontaneously, leads them to believe that dengue fever is an infection of the blood stream with toxic symptoms, involving especially the central nervous system. Their conclusions, they state, are confirmed by the post-mortem pathology of dengue. Certainly, the post-orbital or retrobulbar pain, which they found to be present in seventy-seven per cent. of experimental cases and ninety-three per cent. of spontaneously occurring cases, is a striking feature of the disease. According to them, in frequency of occurrence, it is exceeded only by headaches, and it is at least on a par in diagnostic value with the dermatological signs and adenopathy of dengue fever. It is not unlikely that the retrobulbar pain and soreness are due to a myositis or myalgia of the extrinsic muscles of the globe similar to that which occurs in influenzal infections. When one recalls that other muscles in the body are affected with pain and soreness in dengue fever, it does not seem unreasonable to assume that a similar condition may affect the extrinsic muscles of the globe. This high percentage of occurrence places post-orbital or retrobulbar pain in an important place so far as its diagnostic value in dengue is concerned.

Photophobia is an early symptom in dengue fever and it may precede all other symptoms of the disease. It was present to some degree in practically every patient in the epidemiological group studied, was most marked during the first day or two of the disease, and gradually subsided as the general symptoms became less acute. Most of the patients affected with photophobia suffered acutely, and resorted, of their own accord, to a variety of means to minimize the influence of light upon the eyes.

Conjunctival suffusion or congestion was universally present and was greatest during the first day or two of the disease, subsiding as the other symptoms became less acute. At the height of the congestion the photophobia was most intense, and there was usually quite marked lacrimation. The congestion affected principally the vessels of the bulbar conjunctiva; but, occasionally, those of the palpebral conjunctiva also were affected. Ciliary injection was observed in a number of patients, which, in view of certain other findings, such as post-dengue ciliary weakness and accommodative symptoms, which will be considered in greater detail later in this article, caused one to suspect more strongly that there was present an actual inflammatory involvement of the ciliary body. The fact that the globe was usually sensitive to palpation is additional evidence in this direction.

In a few patients there was an associated discharge of mucus from the conjunctiva, which, however, never attained marked proportions. Eleven of the patients having catarrhal discharge from the conjunctiva were selected for bacteriological investigation. Direct smears were made from the conjunctiva of these patients and examined for microorganisms. All proved to be negative with the exception of two smears in which a few pus cells were found. Cultures were also made from the conjunctival secretion of these patients with negative results in all except two, in which a few colonies of staphylococcus albus appeared on the plates. These studies were abandoned when we failed to isolate any organism which could be regarded as the etiological factor in the production of the conjunctival congestion. The conclusion was reached that the conjunctival congestion and associated discharge were not due to surface action of bacteria, but were, in all probability, due to the effect of a circulating toxin. In this connection it will be recalled that simple hyperemia is in itself sufficient to cause an increase in activity of mucous glands.

A third noteworthy finding referable to the eye in this epidemic, and to which we have been unable to find any reference in the medical literature pertaining to dengue fever, was the constant occurrence of engorgement of the retinal blood vessels. There was some variation in degree, but, in

most instances, it was classified as intense. Its onset was early, the acme being reached on the third or fourth day of the illness, and it subsided in degree from that time until convalescence was established. It was greatest during the period in which the headache and retrobulbar pain were most intense. It was thought that engorgement of the retinal vessels was merely a part of the severe cerebral congestion affecting these patients during the early days of their illness. Support for this belief is furnished by the fact that many of the patients suffered from attacks of epistaxis which caused either a marked amelioration of the headache and other congestive phenomena, or a complete disappearance of these disagreeable symptoms.

In addition to the above findings, some degree of hyperemia of the optic nerve head was present in every patient; but there was no abnormality in its level nor in the sharpness of the margins. It developed concurrently with the engorgement of retinal vessels and was, in all probability, due to the same etiological factors. As the retinal engorgement subsided, the hyperemia of the nerve head decreased and finally disappeared, leaving no residual change. The alteration in color of the nerve head and the retrobulbar pain already referred to strongly suggest the existence of retrobulbar neuritis; but, because of the magnitude of the epidemiological group studied and the discomfort which extended diagnostic procedures would cause these patients, we did not conduct the lengthy examinations required to establish this diagnosis definitely. Such rough tests of the visual fields as we were able to make in studying this group of patients did not reveal defects in the central or peripheral fields, nor alteration in color perception.

Riley and Porter (7) studying a group of fifty-eight patients in the Philippine Islands, found that rachicentesis, with the withdrawal of thirty to forty cubic centimeters of cerebro-spinal fluid resulted in relief of the headaches, retrobulbar pain, and other congestive phenomena in most of their patients. They found that the fluid withdrawn was under markedly increased pressure in eight patients, moderately increased in twenty-nine patients, normal in seventeen and below normal in four patients. They failed to find any cytological or chemical deviation from normal in the fluid. Manometric readings

were not taken, the pressure having been roughly estimated.

Increase in spinal fluid pressure would explain on a mechanical basis the passive congestion of the retinal vessels through increase in the fluid pressure in the vaginal sheath of the optic nerve, forcing the sheath of the nerve outward and causing an angulation of the central vein of the retina, thus impeding the outflow of blood and manifesting itself as an engorgement of the peripheral retinal veins. In view of the fact that no changes in the neuroretinal margin nor in the level of the nerve head were observed, we feel that the increase in cerebro-spinal fluid pressure could not have been very great in any patient of our group, though, in all probability, slight elevation of spinal fluid pressure was the rule. Opportunities were not presented to accurately check the spinal fluid pressure in these patients by rachicentesis and definite proof that elevation in spinal fluid pressure was present is lacking. The writer feels that, in addition to this possible mechanical factor, there is an associated effect due to a circulating toxin, and that the latter is, in all probability, the most important etiological factor in the production of the vascular disturbance, as well as the change in color of the nerve head.

The effect on the retinal as well as on the anterior ciliary and conjunctival vessels may well be due to the direct effect of a circulating toxin on the vessel wall permitting its dilation, though it may be merely a part of a generalized cerebral congestion which likewise may be due to a similar cause. The experiments of Ashburn, Craig, Cleland, et al, already referred to, demonstrate that this is a possibility in that they have all definitely shown that there is a circulating toxin present in the peripheral blood of dengue fever patients.

Another noteworthy phenomenon which was observed in certain patients in this epidemiological group was post-dengue accommodative weakness or insufficiency, which at times became so marked that it could be termed an actual paralysis of the ciliary muscle. The condition usually manifested itself at about the time that convalescence from the fever was established, affected both eyes equally, and

persisted for from two to four weeks. The iris did not share in the paralysis and though there was possibly some sluggishness in response to light stimulation in certain instances, we failed to observe any patient in which there was definite iridoplegia which could be attributed to dengue. Barkan (8) and Van Milligen (9) have both described similar conditions occurring in dengue fever. Couffon and Pagnier (10) have mentioned nearly complete absence of knee jerks and pupillary response to light in a group of dengue fever patients observed by them, and state that the pupillary reflexes returned to normal within a few days after defervescence. Riley and Porter (11) state that they observed a patient in the Philippine Islands in which post-dengue ciliary paralysis persisted for longer than four weeks.

Post-dengue ciliary paralysis or accommodative insufficiency must be differentiated from that due to diphtheria, influenza, focal infections, metabolic disorders such as diabetes, and poisoning by ptomaines.

Many patients suffering with post-dengue accommodative weakness escape detection for several reasons. It will be recalled that the emmetrope, if affected with paralysis of accommodation, is unable to do any near work with the eyes; but so far as distant vision is concerned, little or no effect is produced. In the hypermetrope, paralysis of accommodation makes itself still more noticeable, since, without accommodation, he sees poorly even at the distance; while with the myope, paralysis of accommodation causes little or no inconvenience and the paralysis may be only accidently discovered at the time the eyes are examined. By the same token, presbyopes will not be inconvenienced by paralysis of accommodation if the sclerosis of the lens has reached a certain point and the paralysis of accommodation may, in this instance, remain undetected unless a very careful examination of the eye is made.

When the minimum normal accommodation is less than 1.5 diopters, that is, at about the age of forty-eight, or beyond, it is difficult or even impossible to detect accommodative paralysis. It is therefore evident that a number of patients with paralysis or insufficiency of accommodation following dengue fever will escape detection because of the fact that

they suffer from no inconvenience on account of the paralysis. In this group would be included the myope, the presbyope beyond a certain age, namely, forty-eight years and above; and in addition, we must include individuals suffering only slight discomfort who would ignore their disability altogether. It is estimated that approximately three per cent. of the total number of patients affected with dengue returned because of asthenopic symptoms which were due to ciliary weakness, but, as has already been stated, there is good reason to believe that this number is too small and does not represent the total number affected. Paralysis of the ciliary muscle in dengue is in all probability due to the direct action of a toxin on the muscle.

There were no patients in this series with visual impairment remaining after the paralysis subsided in which the impairment was due to dengue.

Many patients not complaining of accommodative distress stated that viewing motion pictures caused them considerable annoyance and that the effect of bright light was most disagreeable to them. This phenomenon was probably due to a persistent retinal hyper-sensitiveness of toxic origin, made more noticeable to the patient by the bright sunlight usual in sub-tropical zones. It constituted only mild disability, was fleeting in character and disappeared within the first month after the fever.

Neither chromatopsia, paralysis of extrinsic ocular muscles nor keratitis of any type were present in this series of patients.

Bearing in mind that in some epidemics of dengue fever a marked hemorrhagic tendency has been observed, close search was made for evidence of hemorrhage from retinal and choroidal vessels; but there was not a single patient in which this complication was observed. The tendency to hemorrhage was limited entirely to vessels of the nasal mucous membrane and while many of the dengue patients had attacks of epistaxis, it was never profuse. Nosebleed usually resulted very promptly in an amelioration or complete subsidence of many of the disagreeable symptoms such as headache, retrobulbar pain or vomiting, probably because of reduction in blood pressure which, no doubt, influenced indirectly that of the

cerebro-spinal fluid. This phenomenon suggested that venesection would in all probability, relieve many of the disagreeable symptoms, but this therapeutic measure was not tried and we are therefore unable to express an opinion concerning its value in the relief of such symptoms.

Therapeutic measures employed for the relief of subjective eye symptoms were purely symptomatic. Weak astringent lotions in combination with a mild local anæsthetic such as butyn or holocain gave relief from the burning sensation in the eyes. Cool compresses to the lids were especially grateful to the patients and constituted a valuable adjunct to treatment. Subdued lighting and keeping the eyes closed minimized discomfort incident to photophobia and excessive lacrimation. The retrobulbar pain was difficult to influence but was ameliorated to some degree by the internal administration of salicylates and coal tar anodynes.

Post-dengue accommodative weakness required no treatment as it subsided spontaneously in from two to three weeks leaving no residual pathology. The use of suitably tinted lenses is, however, advisable in this group of patients for the protection from glare which they afford.

The use of the eyes for near work should be avoided by patients suffering from accommodative weakness until the ciliary muscle has fully regained its tone. Attention to the general health of the patient during convalescence will minimize discomfort during this stage and hasten the return of the ciliary muscle to its normal state. Insistence upon absolute rest in bed during the acute period of the disease will do much to increase the comfort of the patient and to reduce the incidence of disabling sequelæ.

I am indebted to Major Charles G. Sinclair, Medical Corps, Laboratory Service, and to Capt. Logan M. Weaver, Medical Corps, Eye Department, Station Hospital, Fort Sam Houston, Texas, for the valuable assistance rendered in the investigation of these patients.

Summary and Conclusions.

1. The ocular symptoms of dengue fever such as photophobia, retrobulbar pain, conjunctival congestion, hyperemia of the optic nerve head, and retinal vascular engorgement

constitute important items in the clinical picture of the disease, and are useful as an aid in differential diagnosis.

2. Persistent retinal hypersensitiveness may prove annoying under certain conditions, but it subsides spontaneously and is without harmful after effect.

3. Post-dengue accommodative weakness occurs with sufficient frequency to make it important. It is distressing to the patient while it is present, though the degree of discomfort varies with the individual. It may escape detection in certain patients.

4. Well chosen therapeutic measures can do much to alleviate the distress incident to dengue by minimizing the ocular symptoms.

5. Apparently the unknown toxic agent of dengue has a selective action upon unstriped muscle fibers as evidenced by vasomotor disturbances during the acute stage of the disease and by accommodative disorders following it.

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REPORT OF THE OXFORD OPHTHALMOLOGICAL CONGRESS, 1928.

By B. CRIDLAND, Hon. Secretary.

The XVIIIth Annual Meeting of the Oxford Ophthalmological Congress was held at Oxford on July 5th, 6th and 7th last.

Members met informally at dinner on the 4th in the Hall of Keble College which again offered its hospitality, as in former years. The advantage which is gained by the all too short stay in College where each member is assigned rooms and becomes for the time being a student is quite one of the features of this successful annual gathering of ophthalmic surgeons.

The proceedings of the Congress were held in the Department of Human Anatomy of the University, kindly lent for the purpose by Professor Arthur Thomson, to whom the Congress owes much.

In addition to the Scientific Museum a Commercial Museum was on view in which fourteen well-known firms of ophthalmic instrument makers exhibited the latest forms of apparatus and instruments.

The proceedings commenced on the morning of Thursday, July 5th, with a short address of welcome by the Master, Mr. Philip H. Adams, Reader in Ophthalmology of the University of Oxford.

Immediately following this Professor Leonard Hill opened a Symposium on the Ultra-Violet Ray, to which the following also contributed:—Mr. W. S. Duke-Elder, Dr. W. J. Turrell, Dr. C. M. B. Dobson, F.R.S., Mr. W. R. Ackland, Dr. N. S. Lucas, Mr. G. O. Hume and Mr. A. F. MacCallan; whilst Mr. J. Jameson Evans, Miss Marion Gilchrist and Mr. R. J. Coulter took part in the subsequent discussion.

In his address Professor Hill dealt with the biologically

active rays, their measurement and their penetration of the skin and eye, the theory of the action and immunity to rays, the comparison of sun and lamps as sources, and the action of rays as evidenced by erythema, and touched on hæmo-bactericidal power, rickets, ergosterol, vitamin D and pigment formation.

Some interesting slides of the effect of erythema by ultra-violet rays were shown.

Dr. G. M. B. DOBSON, F.R.S., spoke next on the effect of atmosphere, the altitude of the sun and the presence of ozone on the transmission and the energy of ultra-violet rays.

Curves and photographs illustrating these effects were shown.

Dr. W. J. TURRELL gave his clinical experiences of the practical administration of ultra-violet rays, emphasizing the importance of the regulation of dosage.

The lamp he favored was the Tungsten Arc lamp which he had used for thirteen years.

Mr. W. R. ACKLAND dealt with phototherapy in the treatment of Dental Disease, referring especially to pyorrhœa and its relationship to ocular diseases.

Dr. N. S. LUCAS gave his extremely interesting experiences of the treatment of rickets in Marmoset monkeys by ultra-violet rays, using a mercury-vapor lamp.

An interesting point observed was the effect upon the eyes when exposed to the rays, a reaction came on much more rapidly, *i.e.*, from 1 to 2 hours, than in the human but passed off more quickly. He considered that some ultra-violet ray dosage was essential to monkeys not in their natural state.

Mr. G. O. HUME spoke on the local application of the rays to the eye in certain conditions, emphasizing the importance of selection of cases and dosage.

Mr. A. F. MACCALLAN, C.B.E., whilst recognizing the beneficial effect of phototherapy urged the need for a continued search for casual conditions, notably septic foci in teeth, tonsils, etc.

Owing to illness Mr. W. S. DUKE-ELDER was unable to be present and his paper will appear in company with the full report of the Proceedings of the Congress in the Transactions of the O.S.U.K.

The afternoon was devoted to demonstrations and the examination of ophthalmic apparatus in the scientific and commercial museums.

In the former Mr. E. E. MADDOX showed an Accommodation Balance Test, a new fixation site for the globe in intracapsular cataract extraction and an ingenious apparatus for training amblyopia in squint on a new principle involving hand and eye.

Mr. RAYNER BATTEN showed an interesting collection of fundus drawings of diseases and anomalies of retinal and choroidal vessels.

Dr. MARGARET DOBSON showed a test for astigmatism in which the duochrome principle was applied to the Maddox "V" Test.

Mr. GEORGE YOUNG demonstrated the instruments he now uses in the operation of double sclerectomy for glaucoma which he has devised and also a number of slides in which the effect of the operation could be seen.

Mr. W. NICCOL showed slides of a carcinoma of the choroid secondary to carcinoma of the breast and

Mr. B. CRIDLAND exhibited a test-type for children.

At the conclusion of the afternoon's proceedings members and their friends repaired to the gardens of Trinity College for tea where they were welcomed by Professor and Mrs. R. A. PETERS who kindly acted as host and hostess.

The Annual Dinner of the Congress was held in the evening in the Hall of Keble College and was unusually well attended.

Among the guests were the Regius Professor of Medicine, Sir E. Farquhar Buzzard, K.C.V.O., Professor Arthur Thomson, Miss Kirkaldy, Dr. W. J. Turrell, Dr. C. M. B. Dobson, F.R.S., and Dr. N. S. Lucas.

In speaking on behalf of the Congress the Master, Mr. PHILIP H. ADAMS mentioned that 18 new members had been elected during the year, the total membership being now 414, including Honorary members.

The attendance at the Meeting in 1927 was 104 and included a good number of well known ophthalmic surgeons from the Continent and America.

He regretted to have to record the loss through death of Professor Felix Lagrange, a Foundation Member, Dr. Brown-

low Riddell, Dr. W. J. Tyson, Mr. Fredk. Dale and Mr. T. O'Kelly.

The toast of the visitors was proposed by Mr. R. J. COULTER to which Sir E. FARQUHAR BUZZARD replied.

The proceedings on the morning of Friday the 6th opened with a preliminary report by Mr. BURDON COOPER on "Spectroscopy of the Crystalline Lens" in which he gave with a lantern demonstration the results of the spectroscopic examination of normal and cataractous lenses. The Spectrograms showed that in cataractous lenses potassium was diminished and calcium increased.

Mr. T. HARRISON BUTLER next read a paper on "Sympathetic Ophthalmia" which should be read in the original.

Messrs. S. H. Browning, Malcolm Hepburn, H. M. Traquair and A. W. Ormond joined in the discussion to which Mr. Butler replied.

Then followed the Doyne Memorial Lecture which is undoubtedly the most important contribution to the proceedings of the Congress. Instituted in 1916 in memory of the Founder, the late Robert Walter Doyne, these Lectures have proved the occasion for the appearance of much valuable and original work.

This year the Lecture was by Professor ARTHUR THOMSON, M.A., M.D., F.R.C.S., LL.D., D.C.L., Dr. Lees Professor of Human Anatomy of the University of Oxford, and was no exception to the rule.

Prefacing his remarks with a fitting tribute to the Founder, Professor Thomson chose as his subject "Observations on the Eyes of Birds."

After a review of the literature of the past 250 years on the study of the pecten an exposition of the anatomy of this little understood portion of a bird's eye was given, aided by some excellent slides and models.

It was shown that the most prominent portion of the pecten which projected furthest into the globe was more closely attached to the hyaloid membrane overlying it than it was at its sides.

The hyaloid lying upon this black pigmented structure acted as a dark mirror and the Lecturer suggested that its function might very reasonably be one of elimination of dazzle

from direct rays of the sun with reflection of objects from the upper field of vision, to some other part of the retina.

The value of this in the case of all birds other than prey-birds would be at once seen.

The latter, hovering with his back to the sun in the region of dazzle as far as the ground bird is concerned, is in a position of distinct advantage, whilst the former with his enemy in the region of dazzle and thereby difficult to see is greatly handicapped.

If, however, it is true that the bridge of the pecten is a dark mirror, which can eliminate dazzle and reflect the image of the prey-bird, nature has provided the ground bird with a suitable means of defense.

Some excellent photographs imitating these conditions as far as possible were shown and undoubtedly supported this fascinating and reasonable explanation of the function of the pecten.

The subject is one which has received no attention worth notice since the days of Petit, some 200 years ago, and Professor Thomson is to be congratulated on his work.

The Lecture delivered in a very clear and lucid manner commanded the greatest attention and at its conclusion Professor Thomson was presented with the Doyne Memorial Medal for the year.

Mr. G. R. DE BEER, Lecturer in Comparative and Experimental Embryology in the University of Oxford, then gave a most interesting paper on some original work which he has carried out on the development of the vertebrate eye.

It would be impossible in a few lines to give the essential points brought out and this valuable contribution to the subject should be read in the original.

On the afternoon of Friday, the Scientific and Commercial Museums were again available, but as an alternative a visit to the Morris Motor Works had been arranged and the majority of the members availed themselves of the opportunity to see mass production of motor cars.

On Saturday the 7th the session opened with a speculative paper by Lt.-Col. HENRY SMITH, C.I.E., I.M.S., retd., on the nutrition of the lens and vitreous.

Comparing the uterus with its rhythmic contractions as a

means of maintaining circulation and thereby nutrition he suggested that a sluice-like action of the ciliary muscle of the eye occurred whereby the canal of Schlemm and neighbouring spaces were opened and relaxed and so aided the circulation of the aqueous humor.

Reports of a series of interesting clinical cases were then given by the following:—

Miss Ida Mann, "A Congenital abnormality of the retina"; Mr. John Rowan, "A case of gliomata of the retina" (both eyes removed for gliomata at 1 year and 5 months, no recurrence after over 3½ years); Mr. George Young, "A case for diagnosis" (enuresis up to the age of 17 which disappeared after correction of hypermetropic astigmatism); Mr. J. A. Ross, "Keratitis with intranasal causation" (Keratitis of 15 years standing cured after treatment of an anterior ethmoiditis); Mr. C. G. Russ Wood, "Case of Mikulicz's disease with complications"; Miss Marion Gilchrist, "A case of Keratitis marginalis profunda occurring in both eyes."

All the cases were freely discussed by members.

This concluded the proceedings of as good and pleasant a Meeting as has ever been held.

The attendance was well over a hundred, a matter for congratulation as there were practically no members from abroad owing no doubt to the fact that the International Congress of Ophthalmology will take place at Amsterdam next year.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

A meeting of the Section took place on Friday, June 8th, the chair being occupied by the President of the Section, Mr. Ernest Clarke, C.V.O., F.R.C.S.

Mr. Norman Fleming showed a patient with a congenital anomaly simulating retinitis proliferans. During the past year the right eye had been noticed to diverge painfully; the vision in that eye was $\frac{6}{9}$, that of the left being normal. In the fundus was a marked band of fibrous tissue, stretching from the disc in the lower part of the fundus, and apparently adherent to the fundus. There were also two other bands, and a mass of tissue adherent to each. There was no history of an injury having been sustained, either at birth or since; the birth was a normal one, without instrumentation.

Mr. J. D. M. Cardell showed a case of arborescent cataract. The man, aged 70, had blue-dot cataract and crystalline deposits in the lens. The formation of crystals started from the posterior part of the lens and came forward in the substance of the lens in a tree-like formation.

Mr. Humphry Neame sent a case of irido-cyclitis, which had very fine keratitis punctata on the lower cornea, both right and left; in the right was a complete circle of radiating folds of Descemet's membrane.

Mr. Montague Hine described a case in which an artificial eye burst in the socket and the orbital tissue got sucked into the interior. The case emphasised the fact that there was a negative pressure inside an eye when it was blown while hot, and on the air cooling, a partial vacuum was formed inside.

This woman was cooking, when she felt something explode in her face. She tried to get the artificial eye out, but did not succeed, and the orbit became painful. She went to the ophthalmic hospital, but the house-surgeon there also failed. Mr. Hine then saw the case, and after anæsthetising the patient, he passed a probe to the back of the eye from below, thus displacing the tissue which blocked the hole, and when air was let in, there was an immediate releasing of the eye.

He also showed and described a case of

Neurofibromatosis of the Eyelid.

It was a serious example of the condition, and he came to the conclusion that instead of trying to save the upper lid it would be better to remove the eyelid, eyeball, and some conjunctiva and orbital tissue, and then suture the lids. This was done.

Dr. Harvey Wyatt reported that the under surface of the lid showed chronic inflammation, and this was communicated, by constant contact, to the lower lid, which was ulcerated. Beneath the skin of the upper lid and in the neighborhood of the Meibomian glands were extensive areas of small round-celled infiltration, and a few giant cells. The blood vessels were numerous, very congested and dilated. There was much thickened fibrous tissue from the nerve sheaths. The Meibomian glands contained degenerated and vacuolated cells. In the cornea the epithelium was regular and thin, with somewhat flattened cells; the fibers of the substantia propria were also rather atrophied and thin. Iris and ciliary body showed changes due to chronic inflammation. He was unable to see any hypertrophied end-organs, such as Mr. Treacher Collins described in his case. The disc was cupped.

Mr. Frank Juler congratulated Mr. Hine on the result in his case. He showed pictures of a man whom he exhibited at the Section in 1920. There was then marked neurofibromatosis of orbits and lids and in the temporal and occipital regions. He dissected away much redundant tissue from the eyelids, and the man had been able to carry on until recently. He had now come to have something more done, as there had been a further increase of the neurofibromatous condition. He asked whether Mr. Hine had much hæmorrhage in his case.

In Mr. Juler's own case there was great hæmorrhage, and because of that he did not do so much as he had intended. The patient had buphthalmos in 1920, but as there was vision the speaker did not feel justified in removing the eye. Today there was scarcely any perception of light, and as he was also having some pain in the eye, Mr. Juler thought he ought to advise excision. Today he found, by the corneal microscope, a number of blood vessels spreading from the periphery over the cornea, also a thin line, which might be corneal nerve, or a line of cells, such as Mr. Hine described.

Mr. Treacher Collins described what he found in two cases of similar kind. At the time the cases were exhibited he drew particular attention to the condition of the nerves in the eyeball itself. There were gray streaks in the cornea, a series of elongated cells, which he took to be a new growth around the nerve fibres; it was really connective tissue, not a growth of nerve fibrous tissue. When he examined the buphthalmic eye he found the angle closed, as if it had been an ill-developed angle. He advised removal of the eye in Mr. Juler's case; in that case there was a very deep anterior chamber.

Paper.

Mr. Frederick Ridley, F.R.C.S., read a paper on

Lysozyme: an Antibacterial Body present in great concentration in Tears, and its relation to Infection of the Human Eye.

He said that lysozyme, which was described by Alexander Fleming in 1922, was an enzyme capable of destroying not only non-pathogenic bacteria, but also most of those pathogenic to man and lower animals. It was found in all animal tissues and tissue fluids, and in most secretions. It was present in a high degree of concentration in tears, polymorphonuclear leucocytes, and the nasal mucus and the sputum of man. Fleming showed that if a drop of nasal mucus were placed on the surface of an agar plate, the micrococcus lysodeicticus would not grow in that area, even when thickly planted.

In the pure state, lysozyme was a yellow amorphous powder, easily soluble both in water and saline solution. It was not destroyed by desiccation below 40° C., and retained its potency for many months. Temperatures above 75° C. destroyed it in a short time. It acted efficiently at body temperature, but most rapidly at 60° C. It was quickly destroyed by any considerable quantity of free alkali or acid. It would not pass through parchment or collodion membranes, so it could be purified by dialysis. Wolff, of Holland, showed it gave none of the reactions for protein. It was probably stored in the body by means of fixation by the fats. Following its injection into an animal no anaphylactic phenomena were observed. In high concentrations, lysozyme can kill pathogenic organisms without obvious lysis. The excretions in which lysozyme was not found were cerebro-spinal fluid, urine, and fæces. It was present in turnips, white of egg, fish and fowl. As compared with white of egg, tears had the advantage that they contained only a trace of protein. A fall in the lysozyme concentration of the tears always accompanied epiphora, if that condition had persisted longer than a few hours. In five cases of foreign body retained in the eye more than three days, the concentration in the affected eye was less than 45%, the fellow eye being normal in this respect. In sixteen cases of conjunctivitis, some being acute, some chronic, the infected eye showed a reduction of lysozyme to between 30% and 60%, the other eye in each case usually being normal. In six cases of phlyctenular disease and in four of recent interstitial keratitis, the lysozyme titre of both eyes was below 55% of normal, possibly owing to the associated general ill-health. In five cases of interstitial keratitis which had received thorough general treatment, the titre in both eyes was over 80%.

Concerning the power of lysozyme as an antiseptic, Mr. Ridley showed that one teaspoonful of the pure substance could bestow lethal power on M. lysodeicticus on 100 million gallons of normal saline. The tissues, he said tolerated lysozyme without appreciable reaction in a concentration as great as forty times that found in tears, hence the importance of the substance as an antiseptic was immense.

Dr. Alexander Fleming (the discoverer) spoke briefly in support of the thesis, and showed that the old idea that in-

fection was washed away by secretions, such as tears, was not adequate, and the antiseptic power of these secretions provided the key to the process. The process of inter-cellular digestion of microbes in the process of getting rid of them had not yet been minutely worked out.

MR. LESLIE PATON described the circumstances which led him to induce Mr. Ridley to undertake the research, and spoke of the far-reaching effect of the knowledge on medical work. Col. A. E. Lister and the President also spoke, and Mr. Ridley replied.

REPORT OF THE SECTION OF OPHTHALMOLOGY, NEW YORK ACADEMY OF MEDICINE.

By Dr. THOMAS H. JOHNSON, Secretary.

MEETING MAY 21, 1928. DR. BERNARD SAMUELS, CHAIRMAN.

Dr. John H. Dunnington: A case of **bilateral multiple ocular dermoids** (to be published in full).

Dr. S. Slomka: A case of **essential atrophy of iris.**

Patient, female aged 34 years, was first seen March 14, 1928, and has only complained of headache and poor vision of the right eye for a month, although about 3 years ago she had been told of some peculiar condition of the right eye, but vision in it had been as good as in the other one as recently as one year ago.

Examination: O.D. Vision $\frac{18}{200}$, reduced since to $\frac{6}{200}$. Eye slightly congested. Anterior ciliary veins rather tortuous and engorged. Cornea rather hazy. A.C. normal. Iris very dark brown. Pupil oval and enlarged, about 7*mm* in the long and 5*mm* in the opposite diameter, and displaced temporally and above, upper border about 1*mm* from limbus. At base of iris nasally, above and below, several patches of atrophy, some transilluminous (involving all layers). Vitreous clear. Optic disc pale with moderate cupping at lower temporal border. Rest of fundus normal. Field of vision: concentric contraction. Tension, 32 to 38 (Schiotz).

Dr. Benjamin Friedman: A case of **Kruckenberg's spindle** (to appear in full).

Dr. Harry A. Goalwin read a paper on **sclerosis of the circle of Willis with simple optic nerve atrophy** (with X-ray plates).

A patient of Dr. Arnold Knapp's, a man aged 55 years,

who complains of gradual diminution of vision during the past five years.

His past history is irrelevant except for severe attacks diagnosed as "angina pectoris" six years ago. No attacks during the last five years.

Eye examination: The patient is a myope, wears −12 O.D. and −11 O.S. Vision with this correction $\frac{10}{15}$ O.D. and $\frac{10}{200}$ O.S. The pupils react. There are a few floating vitreous opacities in each eye. The fundus vessels are normal. Both discs show simple optic atrophy. The fields are extremely contracted within five degrees, with preservation of a small, ill-defined island of vision above at the 15° meridian.

Laboratory findings: Blood pressure, 120; Urine examination: negative, Blood Wassermann, negative.

X-ray examination: The skull is normal in shape and size. Its inner surfaces are smooth. The sella turcica is moderately large, but within normal limits. There is evidence of a chronic right frontal sinusitis. The other sinuses are normal.

Both optic canals are normal in shape, size and structure of their walls. Their calculated diameters are: right, 4.3 × 5.1*mm*; left, 3.5 × 4.7*mm*.

THERE IS EVIDENCE OF CALCIFICATION OF THE INTERNAL CAROTID, POSTERIOR CEREBRAL AND POSTERIOR COMMUNICATING ARTERIES.

The calcification of the latter two arteries is distinctly seen on the profile roentgenogram. The posterior cerebral appears as a small annular shadow which is superimposed over the base of the dorsum sellæ. Continuous with this shadow, and running forward toward the site of the internal carotid, is a fine tortuous line. This is the posterior communicating.

The calcification of the internal carotids is seen on the posterior-anterior roentgenogram. It appears as a fine vertical line running from the apex of the petrous pyramid to the lesser wing of the sphenoid. This corresponds to the temporal wall of the artery, as it is situated 5*mm* from the mesial margin of the sphenoidal fissure. A few irregular deposits are seen mesial to this line of calcification.

Since calcification is the final stage in the process of arteriosclerosis, we are justified in considering the above findings as evidence of sclerosis of the circle of Willis.

DIFFERENTIAL DIAGNOSIS: Roentgenologically there are two conditions from which calcification of portions of the circle of Willis should be differentiated.

1. Superimposed shadows of ridges from the temporal bone (squamous portion).

2. Calcification of the various fibrous striæ which normally interconnect the clinoid processes and the apex of the petrous pyramid. The former lines are multiple, curved but not tortuous, each individual line is never parallel to another line within a fraction of a millimeter or even a few millimeters. Oblique projection will displace them from their superimposition on the region of the sella. The latter lines are single and straight, while an artery is usually represented by a double line which is tortuous.

SUMMARY: So far only four such cases have been reported in which the calcification of parts of the circle of Willis were demonstrated roentgenologically during life. (Schuller-Lauber, Horniker.) The condition is undoubtedly much more frequent. It is important to remember that cerebral arteriosclerosis may exist without general arteriosclerosis, that the visual disturbances resulting from it are not necessarily due to pressure of a basal artery on the optic nerve or visual tract.

DISCUSSION, DR. KNAPP: I should like to ask Dr. Goalwin whether calcification of the vessels constituting the circle of Willis is ever accompanied by normal discs. Pressure cannot explain the optic atrophy. How can there be sclerosis of the posterior communicating arteries without involvement of the third nerve?

DR. THOMASSON: How does Dr. Goalwin explain the narrowing of the fields in these cases? May this not be a general arteriosclerosis?

DR. AGATSTON: We must differentiate between sclerosis of the arterioles and the larger vessels. Arteriosclerosis is usually general, while we may have a sclerosis of the large trunks with normal arterioles. Was a Wassermann test made?

DR. GOALWIN: I have never seen a case of sclerosis of the circle of Willis with normal discs.

The irregularity of the field defects is characteristic: some workers have observed peripheral defects, others have recorded

central defects, one a nasal defect and one a bi-temporal hemianopsia.

It is obvious that when sclerosis of a large vessel has occurred there results a disturbance of circulation which will affect the eye even when its arterioles remain normal.

The answer to all these questions becomes easy if we agree with those authors who maintain that eye disturbances are produced not by direct pressure of the sclerosed vessel but by the disturbance of nutrition.

DR. GEORGE S. DIXON: **a demonstration of ophthalmic X-rays.**

Doctor Dixon gave an interesting demonstration from his large experience with X-rays in connection with eye cases.

DISCUSSION: DR. GOALWIN: It would be of great help to the ophthalmic surgeon to become acquainted with the various mechanical processes, such as planing, milling, grinding, etc., because under the same circumstances foreign bodies enter the same eye in the same place and in the same way. The localization of non-metallic foreign bodies was impossible until Vogt introduced his method of bone-free roentgenography by means of a special film which is placed within the conjunctival sac. It would help the surgeon if the localization was reported in terms of coördinates referred to the center of the eyeball, because in that way he could see for himself just what structure of the eyeball lodges the foreign body.

The discussion was continued by Doctors Wheeler, Alger, McDannald and Knapp.

DR. DIXON: Closing, in regard to localization of foreign bodies, I fail to see how taking the center of the globe would be an advantage. We have no trouble in finding foreign bodies in the globe using the indicator on the anterior surface of the cornea. The surgeon wants to know where to cut. We have measured to within *½mm* and that is enough. When a surgeon cuts for a foreign body, his mind goes in a circle while we work on angles.

DR. EDGAR S. THOMSON read a paper on **ocular involvement in sinus disease.**

The author deals with the clinical features of uveitis, retinitis, and optic neuritis caused by sinus disease. Optic

nerve diseases have received ample recognition but uveal diseases, although mentioned in the literature, have not been sufficiently emphasized. They frequently occur in cases in which the sinus symptoms are absent or present only in very low degree, so that it is necessary for the ophthalmologist to decide when the integrity of the eye is sufficiently threatened to justify sinus operation. This is no longer a dangerous procedure in the hands of expert operators. The diagnosis depends on the low-grade, chronic character of the uveal disease, the fact that oftentimes only one eye is involved, the absence of other causes in the general constitution, and, at times, the suggestion of sinus infection in the nose or tonsils. Some of the more important papers of twenty-five years ago are quoted, and a group of papers published within the last eight years. Forty-four cases from the author's private practice are reported briefly.

DR. J. E. MACKENTY: The subject which Dr. Thomson has so admirably presented has always been of great interest to me, as I am convinced of the close relationship between intraocular pathology and sinus disease. I shall confine my remarks to a description of the nasal findings and the methods employed in bringing to light the hidden pathology so characteristic of the concealed or latent type of nasal sinus disease. I shall then say a few words on the palliative and the operative methods used in dealing with this condition.

From a clincial standpoint, one may be permitted three classifications of sinusitis based upon clinical observations:

(a) Purulent sinusitis—the most common type—characterized by a purulent discharge, usually involving all the sinuses. The mucous membrane has a characteristic appearance.

(b) Hyperplastic sinusitis—characterized by frank hyperplastic changes in the mucous membrane, the usual end result being polypi. The discharge is mucoid and mucopurulent.

(c) Concealed, masked or latent sinusitis characterized by very slight or no visible changes in the mucous membrane, vague nasal discomfort or maybe sphenopalatine pain, scanty mucoid or watery postnasal discharge. Microscopic inflammatory changes in the sinus walls, particularly in the posterior ethmoids and sphenoids.

The first and second types are easily detected. It is the third type which arrests our interest, on account of its frequent association with the more serious manifestations of ocular disease and also on account of its insidious nature. Migraine and toxæmia are common manifestations of this type. The detection of this concealed type is no casual task. A most meticulous history of the case must precede a careful and often many times repeated examination of the sinus areas. Careless history-taking perhaps is one of the most usual causes of failure in this field. The history often reveals the inception of the whole trouble as an acute infection, long ago, which left behind it vague yet persistent nasal symptoms. The anterior examination takes note of the color and texture of the nasal mucosa as compared with normal or of the one side as compared with the other. A study of the mucous membrane changes which take place in the choanæ and nasopharynx throws a flood of light upon the condition of the posterior ethmoidal and sphenoidal sinuses. The thin irritating discharge from the posterior sinuses produces the pathological changes observed. On one or both sides of the posterior pharyngeal wall, a thin or thick red line appears. If on one side only, this denotes the side affected. The plica on the septum is thickened, the mucous membrane on the vault of the choanæ may be thick and boggy, or thin and glazed, and irritated looking. Occasionally a little secretion can be seen curving around this edge from the sphenoidal wall. A powerful light is required to appreciate these details as well as much patience and repeated efforts. The X-ray is usually negative. I wish here to *emphasize* as strongly as I may that the final decision as to procedure must always be made by the oculist. The question of palliative or operative treatment should also be decided by him. The rhinologist must, therefore, go no further than to relate his findings. In some cases palliative treatment produces a striking change in the ocular condition. This is strong proof of a causative relationship.

In the concealed type of sinus disease the palliative treatment is as follows:

(1) Cocainization of sphenopalatine ganglia.

(2) Argyrol pack for one hour.

(3) Suction and nasal douche with bicarbonate solution.

(4) The application to the sphenopalatine ganglia area of silver nitrate in increasing strength, starting with 1%.

(5) The saturation of the nasal spaces with ½% carbolized oil.

The operative treatment is as radical as safety, and the experience and skill of the operator will permit. I favor general anæsthesia.

(1) A submucous on the septum is always done to get more room.

(2) One or both sides may be done at the same time, and even a double pansinus with a double Caldwell-Luc have been done at one time when indicated.

(3) Every ethmoid cell is removed.

(4) The sphenoid is widely opened.

(5) The antrum or antra are drained under the inferior turbinate even if no pathology exists in them.

To those who object to this radicalism, I would say:

(1) The results justify it.

(2) The danger of post-operative spreading infection into remaining undrained sinuses (as when a partial operation is done) is obviated.

(3) Secondary hemorrhage is rare since only the base plate and ethmoidal plates remain in the upper nasal regions.

(4) I would put this question—would any of the opponents to this very radical procedure be content, in doing a mastoid operation, to leave any mastoid cells behind? Then, why leave ethmoid cells behind?

I have seen many failures due to conservative sinus surgery.

The after-treatment in sinus surgery is important. Especially is this true in the concealed type of sinus disease. Here we have a chronic osteitis in the remaining sinus walls which tends to continue even after radical removal of the cells. Frequently, the bone hyperplasia seems to be stimulated, for a time, by the operative trauma, and unless controlled, may result in sphenoidal closure and polypoid granulations on the base plate and on the planum. Silver nitrate in increasing strength up to 20% under cocain anæsthesia has a salutary effect locally. Its benefit is also general in controlling toxic absorption. Treatment should

be continued until regeneration of the mucosa over the operative field is complete.

Before concluding, I would make a strong plea for early recognition of the focus, be it in the tonsils, teeth or sinuses, and for early and radical removal of that focus.

DR. E. ROSS FAULKNER: Dr. Thomson's paper has been a great exposition of this very important subject. I would like to see this knowledge propagated throughout the whole medical profession, as it seems to me that very few have grasped the significance of the correlation between the obscure head infections and the serious lesions which affect the eyes. To illustrate this, I would like to refer to the case of a child which was brought to me with double optic neuritis after a very extensive investigation by well-known physicians who had finally made a diagnosis of a brain tumor. Dr. Thomson saw the patient, and stated he was very suspicious that the eye condition came from the sinuses. This was in June, 1926. The child was 13 years old, had had influenza in February and the eyes began giving trouble in April, first the left eye then the right. At the time I saw her, RV: $\frac{11}{11}$, LV: $\frac{10}{100}$, with an optic neuritis on both sides. I opened her sinuses on both sides and the next day the right eye was normal; two days later the left eye was normal.

Another case I wish to mention to illustrate how difficult the diagnosis may be and how fine a thread one may hold to turn the balance of evidence, and in this case it largely depended on the history. The patient had lost his left eye in childhood and for several months had failing vision in the right eye with frequent hemorrhages into the macular region. During this time he had been investigated by excellent specialists and the sinuses pronounced negative. The tonsils had been removed and the teeth found negative. He gave a history of an antrum infection 15 years before which had lasted six weeks and had been punctured twice a week and washed out during that period till it was finally pronounced cured and had not troubled him since. Washing out the antrum the fluid returned clear, a fact which I corroborated with others who had examined him. I was persuaded, however, that the trouble came from the antrum and asked him to have an opening made in the inferior meatus.

This was done and the antrum irrigated frequently with no result. He had night sweats and seemed toxic during the first two or three weeks after the operation. I felt that I had only stirred up a focus which should be removed thoroughly by a radical operation. His eye showed no improvement and he went away. I did not see him for a year when he was sent back to me by a specialist in Philadelphia for a radical antrum operation as I had previously advised. On opening the antrum I found on the floor a dirty mass of granulation tissue ½ inch thick containing flakes of inspissated pus. I cleaned this all out and he made a very rapid recovery in his eye symptoms, also his general health began to improve. The eye continued to improve till he got $\frac{11}{8}$ vision. This case verifies a contention which I have had, viz., that these head infections can remain latent for long periods just as syphilis may, and often show as little evidence of the initial invasion as syphilis, and that the history may be the most important part of the evidence.

DR. WHEELER: We are indebted to Dr. Thomson for a comprehensive treatise on an important subject. The essayist has viewed his subject broadly, and has contributed a valuable review of the literature. Dr. Thomson has pointed out especially that an inflammatory process in the paranasal sinuses may be responsible for not only retrobulbar neuritis but also for inflammatory reactions in the uveal tract. I should like to ask Dr. Thomson whether he believes that after careful examination of the affected eye he can say that probably accessory sinus disease is or is not responsible.

There is a rare but important condition that concerns us in considering the subject under discussion that is not mentioned in the paper. I refer to mucocele of the sphenoidal sinus. In this condition the opening into the sinus is non-existent, sometimes probably congenitally so. The result is in effect a retention cyst, and the products of cell life in the mucous membrane lining collect, and the mucocele expands indefinitely, encroaching on the surrounding vital structures. The most important of these are the optic nerves. Complete atrophy of one optic nerve and partial atrophy of the other have resulted in several cases. I have had one such case. When exploratory puncture of the sphenoid was made a

quantity of brown non-purulent fluid gushed forth. In the laboratory examination of the fluid the striking finding was a large number of cholesterin crystals. Clinically mucocele of the sphenoid results in proptosis, impairment of motility of the eye or eyes, dilatation of the pupil, impairment of sight from optic atrophy. X-ray examination shows dilatation of the sinus and loss of detail of body structures at the apex of the orbit. In 1922 van de Hoeve reported a case and reviewed eight other cases from literature. He brought out the impressive fact that in not one of the cases was the proper diagnosis of mucocele of the sphenoidal sinus made before operation. In thinking of ocular manifestations of sinus disease let us not forget the possibility of this rare, destructive condition and diagnose it, if possible, before operation.

DR. KNAPP was very much interested in Dr. Thomson's statement that the occult form of sinusitis was a very definite factor in causing uveitis. Its importance in optic nerve lesions is generally accepted. Has Dr. Thomson any opinion on the relative frequency of the latent and manifest sinus cases in causing uveal disease?

DR. THOMSON in reply to Dr. Knapp said he had no definite figures on percentages to give but was under the impression that uveitis occurred more commonly from the occult sinus cases than from the frank ones. Answering Dr. Wheeler, he said he had not seen many cases of mucocele and could not add to Dr. Wheeler's description. Iritis from sinus disease has no special characteristics. It is a low-grade plastic inflammation which does not differ from the iritis caused by other conditions. The diagnosis must rest on its unilateral character, and the absence of other constitutional causes. Also the history of a recurrent condition is suggestive. Confirmatory symptoms in the sinuses are helpful when present.

DR. MARTIN COHEN demonstrated **a fundus colorimeter.** He said:

We have long felt the need of an instrument for recording the increasing or decreasing pallor or redness of the optic disc or other fundus areas. This instrument I devised to serve as a clinical aid in the diagnosis and prognosis of such conditions as retro-bulbar neuritis, multiple sclerosis, tabes, brain affections, accessory sinus diseases, slight color dif-

ferences between the optic discs, and other pathological processes. Our judgment of color changes depends upon our visual impression of previous ophthalmoscopic examinations which are difficult or impossible at times to recall. We also infer color alterations from examination of the visual acuity and perimetric tests; these latter tests are of subjective value and are frequently unreliable owing to the patient's poor vision or lack of coöperation. Whether this instrument will be able to detect color changes earlier than the functional tests can not yet be stated, owing to the fact that clinical study in this respect has not been completed.

Two years ago I started to develop this instrument and after careful consideration decided upon a colorimetric principle to be applied in its construction. Finally I concluded to use a calibrated red circular wedge to be attached to the electric hand ophthalmoscope.

The construction of the wedge was supervised by Dr. Jones at the Research Laboratory of the Eastman Kodak Company. It consists of a gelatin filter stained with eosin; the wedge has a clear color near its apex, then takes on a faint reddish tint which gradually becomes a deep red color towards its base. It is 10*mm* in diameter, thin, and sealed with Canada balsam between cover glasses. After the wedge was completed it was sent to Mr. Poser of the Bausch & Lomb Optical Company, who had it attached to the electric hand ophthalmoscope, the instrument thus serving as an electric ophthalmoscope and colorimeter.

A narrow, thin, graduated scale surrounds the circular wedge; it reads from zero at its apex to 180 degrees at its base. The wedge with its scale is attached to the ophthalmoscope and to a rod having at its base a wheel which is readily turned, producing the revolution of the graduated wedge. The light from the electric bulb of the ophthalmoscope passes upward through a circular aperture upon which is placed the graduated revolving circular wedge. The rays of light, after entering the aperture, come in contact with the surface of the total reflecting prism whence the rays pass into the patient's eye, to be reflected back into the examining eye.

The light rays which enter the patient's fundus through the clear portion of the wedge appear as a whitish disc, similar in

size to the normal optic disc. The whitish disc which is projected into the patient's eye is used for fundus examinations. If color changes in the optic disc or elsewhere are to be studied, the colorimeter is brought into play by gently revolving the wheel which is conveniently located near the handle of the ophthalmoscope. This procedure gradually changes the color of the whitish projected disc on the fundus to gradations of red. As soon as the white disc appears distinctly red the reading, which is readily seen, is noted; this notation is used for comparison in future examinations of the case.

To conduct the colorimetric examination, it is necessary to dilate the pupils, to have the refraction properly corrected by lenses in the ophthalmoscope. The illumination from the ophthalmoscope should be of the same brightness at each subsequent examination; this can be readily recognized and practically determined by observation.

It is well known that the color of the disc and other areas is not a criterion of pathology, but if alterations increase or decrease in these areas they are usually due to pathological conditions.

The colorimetric test just described is of value only in individual cases when conducted by the same examiner, for the reason that color differences and impressions vary in different individuals; therefore a definite comparison is essential.

At a future meeting I shall report on the results obtained with the use of the fundus colorimeter in certain clinical cases.

INDEX OF AUTHORS AND SUBJECTS.

VOL. LVII.

Abstracts, Book Reviews and Notices, 104, 310, 316, 325, 553
"Acta Ophthalmologica," Notice of, 107
ADLER, FRANCIS HEED, A Comparative Study of the Rôle of Pigment in the Physiology of Vision, 346
AGATSTON, S. A., Fundus as Definite Index to Arterial Disease, 100 Cases, 386
Animal and Human Eyes, 531
Aqueous Humor, The Experimental Transmission of Arsenic to the, (A. M. Yudkin, A. C. Krause and D. G. Morton) 147
Arsphenamines, Rôle of the, in the Production of Ocular Lesions, (E. L. Zimmermann) 509
Artificial Eye, Bursting of, 645
ASBURY, MARY KNIGHT, A Pathological Study of Two Cases of Ocular Tuberculosis, 603
ATKINSON, WALTER S., Two Safety Measures in Cataract Extraction, 275

BARKAN, HANS, Air Embolism of the Retinal Vessels, 402
BARKAN, OTTO, Cloquet's Canal, Visible in the Living, with Observations of Hemorrhage into Cloquet's Canal, 502
BEDELL, ARTHUR J., Photographic History of a Traumatic Retino-Choroiditis, 262
BERGHAUSEN, OSCAR, Tuberculin Therapy in Ocular Tuberculosis, 583
BIETTI, A., "Trattato di Oftalmojatria" (Handbook of Ophthalmology), Review of, 320
Birds, Eyes of, (Arthur Thomson) 642
Blepharoptosis, Modified Motais Operation for, (D. B. Kirby) 227
Blind, Treatment of the Adult, in Germany, (Betty Hirsch) 178
BROWN, ALBERT L., Rapid Dilation of Lacrimal Sac with Sea Tangle Probes, 397
BROWN, E. V. L., The Division of Ophthalmology in the New University of Chicago School of Medicine, 190
BURKY, EARL L., and WOODS, ALAN C., Lens Protein—New Methods for the Preparation of Beta Crystallin, 41
BURKY, EARL L., and WOODS, ALAN C., Lens Protein—The Isolation of a Third (Gamma) Crystallin, 464
BUTLER, T. HARRISON, "An Illustrated Guide to the Slit-Lamp," Notice of, 215

Cancers of Orbito-Palpebral Region Treated with Radiation, (George T. Pack) 246
Case Reports, Minor, of Interest, 196
CASSELL, JAMES WILSON, The Eye Clinic of Dr. Henry T. Holland, at Shikârpur, India, 380
Cataract, Non-Operative Treatment of, (Zenas H. Ellis) 46; Operation for Shrunken, in Adolescents, (Arnold Knapp) 594; Proteins of the Lens and their Chemical Changes in the Production of Senile, (I. S. Tassman) 361
Cataract-Extraction, Two Safety Measures in, (Walter S. Atkinson) 275
CHAMBERS, STANLEY O., Review of Literature of the Treatment of Syphilitic Eye Diseases, 412

Chloroma, with Ocular Lesions, (Martin Cohen) 238, 303
Choked Discs, A Study of Recession of, Following Operations for Brain Tumor, (Gilbert Horrax and Cameron Haight) 467
Choroid, Hemangioma of the (S. B. Marlow) 484
CHOU, C. H., A Typical Form of Familial Degeneration of Cornea (Fleischer), 574
Ciliary Body, Melanotic Sarcoma of, 195; Sarcoma of, (Arnold Knapp) 302
Cloquet's Canal, Visible in the Living, with Observations of Hemorrhage into Cloquet's Canal, (Otto Barkan) 502
COHEN, MARTIN, A Case of Chloroma with Ocular Lesions, 238
COHEN, MARTIN, KILLIAN, JOHN A., and KAMNER, MILDRED, Comparative Chemical Studies of the Ocular Fluids of Cerebrospinal Fluid, and of the Blood, 59
Colorimeter, (Martin Cohen) 659
Cornea, Typical Form of Familial Degeneration of (Fleischer), (CHOU, C. H.) 574
COWAN, ALFRED, Test Cards for the Determination of the Visual Acuity, 283
CUSHING, HARVEY, and BAILEY, PERCIVAL, Hemangioma of Cerebellum and Retina (Lindau's Disease), with Report of a Case, 447
Cyclophoria, Ames Spectacle Device for Treatment of, with Successful Case, (W. B. Lancaster) 322

Dengue, Ocular Symptomatology in, (William D. Gill) 269
DERBY, GEORGE S., The Nature of the So-Called Koeppe Nodules, 561
DUKE-ELDER, W. STEWART, Intra-Ocular Fluids, 100; Recent Advances in Ophthalmology, 210

ELLER, J., The Significance of the Ciliary Epithelium in the Absorption of Vitreous Hemorrhages, 134
ELLIS, ZENAS H., Non-Operative Treatment of Cataract, with Report on Lens-Antigen Treatment, 46
Ephedrine, Experience with, in Ophthalmic Practice, (Mark J. Schoenberg) 272
Eye, Diseases of the, and Adnexa Due to Fungi and Higher Bacteria, (S. R. Gifford) 224
Eye, Two Early American Works on the, (George H. Jackson, Jr.) 265
Eyelashes, in Iris, 546
Eyelids, Collodion to Keep the, Closed after Infiltration Anæsthesia, (William D. Gill) 269; Neurofibroma of, 646
Eye Strain in Relation to Functional Neurosis, (F. W. Marlow), 339
Eyes, Pathological Pictures of the, 545

FUCHS, ADALBERT, "Atlas of the Pathology of the Eye," Notice of, 106
Fundus, Analysis of 100 Cases of Fundus Examinations as Index of Arterial Disease, (S. A. Agatston) 386

GIFFORD, S. R., Diseases of the Eye and Adnexa Due to Fungi and the Higher Bacteria, 224; Some Modern Preparations Used in the Treatment of Glaucoma, 612
GILL, WILLIAM D., Ocular Symptomatology in Dengue, 628; A Way to Keep the Eyelids Closed after Infiltration Anæsthesia, 269
Glaucoma, in Generalized Vascular Nævus of the Skin. Report of a Case with Angiomatous Changes in the Iris, (Arnold Knapp) 219; Some Modern Preparations Used in the Treatment of, (S. R. Gifford) 612; Temporary and Permanent Disturbances of Dark Adaptation in, 550; The Non-Operative Treatment of Chronic, (Searle B. Marlow) 165; Value of Scotometry in the Diagnosis and Treatment of, (A. H. Thomasson) 160

HECHT, SELIG, Visual Acuity and Illumination, 564
HECKEL, EDWARD B., Diphtheria Antitoxin in Large Doses for Sympathetic Ophthalmia, 54
HIRSCH, BETTY, Training of the Adult Blind in Germany, 178, 206
HORRAX, GILBERT, and HAIGHT, CAMERON, A Study of the Recession of Choked Discs Following Operations for Brain Tumor, 467

Hyperphoria, Operation for, 194
Hyphæma, Recurrent, in Anterior Chamber from a Tumor, 299

India, Eye Clinic of Dr. Holland at Shikarpur, (James Wilson Cassell) 380
Intra-ocular Pressure, 299; Effects of Illumination upon, 550
Iridocyclitis with Increased Tension, Discussion on, 533
Iris, Essential Atrophy of, (Slomka) 650

JACKSON, GEORGE H., JR., Notes on "Two Early American Works on the Eye," 265
JOHNSON, GEORGE LINDSAY, "Contributions to the Comparative Anatomy of the Reptilian and the Amphibian Eye," Notice of, 553

Keratitis, Relapsing, 196
Keratoconus, Report of Three Cases of, (Ervin Török and L. D. Redway) 19
KIRBY, DANIEL B., Modified Motais Operation for Blepharoptosis, 327
KNAPP, ARNOLD, Glaucoma in Generalized Vascular Nævus of the Skin. Report of a Case with Angiomatous Changes in the Iris, 219; On the Operation for Shrunken Cataracts in Adolescents, 594
Koeppe Nodules, Nature of the So-Called, (G. S. Derby) 561
KOFLER, KARL, and UNBANEK, JOSEF, "Vereinfachung und Verbesserung der West-Polakschen Traenen sackoperation," Notice of, 310

Lacrimal Sac, Rapid Dilation with Sea Tangle Probes, (Albert L. Brown), 397
Lagrange Operation, Lantern Slides of, 547
LAMB, H. D., Myxoma of the Orbit, Anatomical Findings, 425
LANCASTER, WALTER B., Ames Spectacle Device for Treatment of Cyclophoria. Report of a Successful Case, 332
Lens Antigen, Treatment of Cataract with, (Zenas H. Ellis) 46
Lens deformities, 196
Lens Protein: Isolation of a Third (Gamma) Crystallin, (Earl L. Burky and Alan C. Woods) 464; New Methods for Preparation of Beta Crystallin, (Earl L. Burky and Alan C. Woods) 41
Lenticonus Posterior, (Henry H. Tyson) 38
Leukæmia, Eye Conditions in, (Benjamin Sachs) 474
Lid, Melanoma of Margin of, 195
Lids and Conjunctiva, Melanosis of, 194
Lysozyme in the Tears, 647

Macular Artery, Obstruction of, 196
MARLOW, F. W., Eye Strain in Relation to Functional Neurosis, 339
MARLOW, SEARLE B., A Case of Hemangioma of the Choroid, 484; The Non-Operative Treatment of Chronic Glaucoma, 165
MEEKER, LOUISE H., and COOK, WARD H., Methods of Preparing Gross and Microscopic Eye Specimens, 185, 206
MEESMANN, "Die Mikroskopie des Lebenden Auges," (Microscopy of the Living Eye: Slit Lamp Atlas), Review of, 317
MEYER, S. J., Detachment of the Zonular Lamella, 121

NEAME, HUMPHREY, and WILLIAMSON-NOBLE, F. A., "A Handbook of Ophthalmology," Notice of, 216
NEWCOMB, MAJOR C., and WRIGHT, R. E., LT. COL., Some Analytical Observations on the Vitreous Humor from Normal and Glaucomatous Eyes, 460
New York Academy of Medicine, Report of the Ophthalmological Section of the, 87, 204, 325, 430, 543, 650

OAST, SAMUEL P., Blue Sclerotics and Brittle Bones, Occurrence of, in Mother and Child, 254
Ocular Fluids, Cerebro-spinal Fluid, and Blood; Comparative Chemical Studies of the, (Martin Cohen, John A. Killian, and Mildred Kamner) 59
Ocular Tuberculosis, Pathologic l Study of Two Cases of, (Ma y Knight Asbury) 603
Ophthalmological Congress, Amsterdam, 1929, 218, 444

Ophthalmology, Historical Perspectives in, 308
Ophthalmology, Report on the Progress of, 91
Ophthalmology, The Division of, in the New University of Chicago School of Medicine, (E. V. L. Brown) 190
Optic Atrophy in Sclerosis of the Circle of Willis, 650
Optic Disc, Isolated Arteries of the, 546
Optic Nerve, A Case of Glioma of the, (Herbert Wright Wootton) 377
Optic Neuritis, Discussion on, at British Medical Association, 199
Optical Phenomena, Stage for Demonstration of, 549
Orbit, Discussion of Inflammatory Affection of the, 537; Myxoma of the, Case of, and Anatomical Findings, (H. D. Lamb) 430
Orbito-palpebral Region, Treatment of Cancers of the, by Radium, (George T. Pack) 246
Oxford Ophthalmological Congress, Report of, 72, 639

PACK, GEORGE T., Radiation Therapy of Cancers of the Orbito-palpebral Region, at the Radium Institute of the University of Paris, 246
Papiloedema, Bilateral, in Brain Tumor, 298
Paralysis, Traumatic Ocular, with Retraction Movements, 204
PETER, LUTHER C., "The Extra-Ocular Muscles," Review of, 322
Protein Therapy, Specific and Non-Specific, in Ophthalmology, (Alan C. Woods) 488

Refraction, One Hundred Cases of, 540
Retina, Air Embolism of the Vessels of the, (Hans Barkan) 402; Angioid Streaks in the, 547
Retina and Cerebellum, Report of a Case of Hemangioma of (Lindau's Disease), (Harvey Cushing and Percival Bailey) 447
Retina and Optic Tract, Reports on, 91
Retinal Neoplasms, Natural History of, 532
Retino-choroiditis, Photographic History of a Traumatic, (Arthur J. Bedell) 262
Royal Society of Medicine, Report of the Ophthalmological Section of the, 81, 194, 298, 441, 645

SACHS, BENJAMIN, Eye Conditions in Leukæmia, 474
SCHOENBERG, MARK J., Experience with Ephedrine in Ophthalmic Practice, 272
Sclerotics, Blue, and Brittle Bones, Report of Occurrence of, in Mother and Child, (Samuel P. Oast) 254, 304
Sinus Disease, Ocular Involvement in, (Edgar S. Thompson) 653
Slit-Lamp, attachments, 307
SLUDER, GREENFIELD, "Nasal Neurology, Headaches and Eye Disorders," Review of, 316
Squint, Recession without Tenotomy, 544
Sympathetic Ophthalmia, Large Doses of Diphtheria Antitoxin in Treatment of, (Edward B. Heckel) 54
Syphilitic Eye Disease, Literature of, (Stanley O. Chambers) 412

TASSMAN, I. S., The Proteins of the Lens and their Chemical Changes in the Pathogenesis of Senile Cataract, 361
Test Cards for Determination of the Visual Acuity, (Alfred Cowan) 283
Third Nerve Paralysis, Correction of Defect Due to, (Meyer Wiener) 597
THOMASSON, A. H., The Value of Scotometry in the Diagnosis and Treatment of Glaucoma, 160
TÖRÖK, ERVIN, and REDWAY, L. D., Three Cases of Keratoconus, 19
TRAQUAIR, H. M., "An Introduction to Clinical Perimetry," Notice of, 104
Tuberculin Therapy in Ocular Tuberculosis, (Oscar Berghausen) 583
Tuberculosis, Clinical Aspects of Ocular, (W. H. Wilmer) 1
TYSON, HENRY H., Lenticonus Posterior, 38

United Kingdom, Ophthalmological Society of the, 531
Uveal Tract, Bilateral Sarcoma of the, 543

Vision, Study of the Rôle of Pigment in the Physiology of, (Francis Heed Adler) 346
Visual Acuity and Illumination, (Selig Hecht) 564
Vitiligo, Ocular Depigmentation with Generalized, (David Wexler) 393
Vitreous, Curative Value of Aspiration of the, (Zur Nedden) 109
Vitreous Hemorrhage, Recurrent, 304
Vitreous Hemorrhages, the Significance of the Ciliary Epithelium in the Absorption of, (J. Meller) 134
Vitreous Humor, Some Analytical Observations on the, from Normal and Glaucomatous Eyes, (Major C. Newcomb and Lt. Col. R. E. Wright) 480

WEXLER, DAVID, Ocular Depigmentation Accompanying Generalized Vitiligo, 393
WIENER, MEYER, Correction of Defect Due to Third Nerve Paralysis, 597
WILMER, W. H., Clinical Aspects of Ocular Tuberculosis, 1
WOODS, ALAN C., Protein Therapy, Specific and Non-Specific, in Ophthalmology, 488
WOOTTON, HERBERT WRIGHT, A Case of Glioma of the Optic Nerve, 377

X-Rays, Ophthalmic, 653

YUDKIN, A. M., KRAUSE, A. C., and MORTON, D. G., The Experimental Transmission of Arsenic to the Aqueous Humor, 147

ZIMMERMANN, E. L., The Rôle of the Arsphenamines in the Production of Ocular Lesions, 509
Zonular Lamella, Detachment of the, (S. J. Meyer) 121
ZUR NEDDEN, The Curative Value of Aspiration of the Vitreous, 109

GENERAL INDEX.[1]

VOLUMES XLIII TO LVII.

BY DR. JAMES A. SPALDING, PORTLAND, ME.

Abducens Nerve, Variations in, (T. B. Magath) xlviii, 67
Abiotrophy, Ophthalmoplegia Externa, lii, 56
Accommodation, Anomalies of, (A. Duane) xlv, 124; Apparatus, (Harvey J. Howard) xlix, 182; New Theory of, (G. Lindsay Johnson) liii, 426; Normal Reserve of, in Presbyopia, (F. J. Slataper) lv, 320; Persistent Spasm of, from Latent Hyperphoria, (F. W. Marlow) li, 223; Spasm of, (J. W. White) xlvii, 466; Subnormal, (A. Duane) liv, 566
"Acta Ophthalmologica," lvii, 107
ADLER, FRANCIS HEED, A Comparative Study of the Rôle of Pigment in the Physiology of Vision, lvii, 346; Action of Atropine in Ocular Inflammation, lv, 484; Eye Disorders in Deficiency Diseases, lvi, 593; Local Control of Ocular Circulation, liii, 1; Pupilloscopic Findings in Lesions of the Reflex Arc, lv, 262
ADLER, FRANCIS H., and LANDIS, E. M., Effect of Miotics and Mydriatics on Protein Contents of Aqueous, liv, 265
ADLER, FRANCIS HEED, LANDIS, E. M., and JACKSON, C. L., Tonic Effect of Sympathetic on Ocular Blood Vessels, liii, 239
Adrenalin in the Glaucomatous Eye, (Arnold Knapp) l, 556
After-Cataract and Immature Cataract Operations, (T. Heyl) xlvi, 53
AGATSTON, S. A., Fundus as Definite Index to Arterial Disease, lvii, 386
ALGER, ELLICE M., Therapeutic Suspension of the Fusion Impulse, lv, 439
ALLING, ARTHUR N., Exfoliation of Lens Capsule, lvi, 1; Fat Transplantation after Enucleation, xlviii, 263
Amaurosis, Retrobulbar, Foreign Protein Treatment, (Julius Wolff and Joseph H. Globus) lvi, 302, 576
Amaurotic Family Idiocy, (Martin Cohen) lii, 149
Anæsthesia, General, in Ophthalmic Practice, (James T. Gwathmey) liii, 579
Anaphylaxis, Ocular, (A. C. Woods) xlvi, 8, 283, 503; Ocular, Reaction of Perfusion with Specific Antigen, (Alan C. Woods) xlv, 557
ANDREWS, J. A., Anterior Chamber Syringe, xlvi, 256; Electrical Device for Illumination of Operations, xlviii, 65
Anilin Injuries of Eye, (R. J. Curdy) xlv, 243
Anisocoria, Loss of Correlation Between the Eyes, (Anton Lutz) xlvii, 587

[1] Students interested in the minutiæ of ophthalmological history may find closer details in the indexes of the separate volumes year by year. This index includes authors and papers in full. Reports of meetings and of brief cases can be found in the yearly index attached to each volume.

Anterior Chamber, Traumatic Transplantation of Cilia into, (H. L. Begle) xlvi, 47
Antidiphtheritic Serum in Hypopyon Keratitis, (Ben Witt Key) xlviii, 527
Anti-Typhoid Vaccine, Keratitis Following Inoculation with, (G. H. Bell) xlviii, 273, 368
Aqueous, Effect of Mydriatics and Myotics on Protein Contents of, (F. H. Adler and E. M. Landis), liv, 265
Aqueous Humor, The Experimental Transmission of Arsenic into the, (A. M. Yudkin, A. C. Krause, and D. G. Morton) lvii, 147
Argyll-Robertson Pupil, (John Dunn), xlvii, 469
Arsphenamines, Rôle of the, in the Production of Ocular Lesions, (E. L. Zimmermann) lvii, 509
Arteriosclerosis, Scientific Treatment with Iodide of Potash, (F. Flury) xliii, 393
Artificial Eyes in Enucleation Substitutes, (P. Gouglemann) xlviii, 268
ASBURY, MARY KNIGHT, Pathological Study of Ocular Tuberculosis, lvii, 603
Asphyxia, The Eye in Traumatic, (Paul D. Berrisford), l, 411
Asthenopia, Weak Atropine Solutions in, (William E. Gamble) lii, 160
Astigmatic Axis, Law of Rotation of, (A. W. Stirling) lii, 63
Astigmatism, Influence of Age on the Axis of, (A. W. Stirling) l, 19
ATKINSON, WALTER S., Two Safety Measures in Cataract Extraction, lvii, 275
Atropine in Ocular Inflammation, (Francis Heed Adler) lv, 484
Aviators' Eyes, (W. H. Wilmer) xlviii, 439
AXENFELD, T., "Die Aetiologie des Trachoms" (Causes of Trachoma), Notice of, xliv, 360; "Lehrbuch der Augenheilkunde," xlv, 106
AYER, JAMES B., Lumbar Puncture and Spinal Fluid in Eye Diseases, xlv, 9

BALBUENA, FELIX FERNANDEZ, Reduced Silver Nitrate for Section of Retina, lii, 358
BALL, JAMES MOORES, and LAMB, HARVEY D., Epibulbar Melanotic Sarcoma Sixteen Years after an Injury, lii, 80
BARKAN, HANS, Air Embolism of the Retinal Vessels, lvii, 402; Industrial Trauma and the Development of Ocular Tuberculosis, Syphilis and Neoplasms, li, 103
BARKAN, OTTO, Cloquet's Canal, Visible in Life, with Hemorrhage into It, lvii, 502; Differential Pupilloscopy, li, 29
BARLOW, AARON, Fundus Changes with or without Nephritis, lvi, 356
BARNERT, CYRIL, Chloroma of Orbit, liii, 454
BARRAQUER, IGNACIO, Phakœresis, l, 307, 448, 501
Barraquer's Extraction, Eighty-seven Cases of, (R. Afleck Greeves and R. Foster Moore) lii, 386
Barraquer, Working with, (William McLean) lii, 460
Basedow's Disease, Tetany and Acromegaly, (Schoenborn) xliv, 421
BEACH, S. J., Familial Maculocerebral Degeneration, lvi, 286
BEARD, C. H., "Ophthalmic Surgery," Notice of, xliii, 466
BECK, JOSEPH, "Applied Pathology in Diseases of Nose, Throat and Ear," liii, 408
BEDELL, ARTHUR J., Photographic History of a Traumatic Retino-Choroiditis, lvii, 262; Traumatic Pulsating Exophthalmos, xliv, 139
BEDNARZKI, A., Decompression in Optic Nerve Diseases, xliv, 53
BEGLE, H. L., Blood Staining of the Cornea, xliii, 393; Traumatic Transplantation of Cilia into Anterior Chamber, xlvi, 47
BELL, GEORGE HUSTON, Bilateral Papilloedema from Sinuses, xlvii, 344, 422; Keratitis Following Anti-Typhoid Vaccine, xlviii, 273; Sarcoma of Iris, xlvi, 427; 1260 Cases of Prevention of Post-Operative Intraocular Infections, lii, 436
BELL, GEORGE H., and TOUSEY, SINCLAIR, Orbital Tumor Cured with Radium, xlviii, 531
Bell's Phenomenon, Pathology of, (E. Kraupa) xliv, 281
BENEDICT, WILLIAM L., Iritis, and Focal Infection, l, 560

BENEDICT, WILLIAM L., and KNIGHT, MARY S., Inflammatory Pseudo-Tumor of Orbit, lii, 582
BENEDICT, WILLIAM L., VON LACKUM, WILLIAM H., and NICKEL, ALLEN A. C., Pelvic Organ Infection in Eye Diseases, lvi, 116
BERENS, CONRAD, JR., Eye Clinic in Penal Institutions, xlvii, 448; Out Patient Ophthalmology, li, 519
BERENS, CONRAD, JR., and STURGES, GERTRUDE, Case Records, lii, 259
BERGHAUSEN, OSCAR, Tuberculin Therapy in Ocular Tuberculosis, lvii, 583
BERGMEISTER, R., "Die Tuberkuloesen Erkrankungen des Auges," lvi, 527
BERRISFORD, PAUL D., Annular, Contusion, Lens-Opacity, xlvii, 616; Eye Findings in Traumatic Asphyxia, l, 411
BEST, HARRY, "The Blind," xlix, 549
BIETTI, "Trattato di Oftalmojatria," lvii, 320
BIRKHAEUSER, R., "Scalæ Typographicæ," li, 614
BISTOS, J., Causes of Heterochromia, xliv, 433; Investigations on the Ætiology of Heterochromia, xliv, 433
BLAAUW, E. E., Circumscribed Lens Changes, liv, 305; "Eye Symptoms of Reynaud's Disease," xliii, 683
BLACK, NELSON M., and HAESSLER, F. H., Dystrophic Intracellular Corneal Epithelium Opacity, lvi, 151
BLAKE, EUGENE M., Allergic Reaction of Eyelids, liii, 272; Bilateral Detachment of Retina in Pregnancy Nephritis, and Reattachment, li, 586; Congenital Hydrophthalmus, liv, 1; Evulsion of Optic Nerve, xlvii, 567
BLANTON, W. B., "A Manual of Normal Physical Signs," lvi, 314
Blepharochalasis, (F. H. Verhoeff and Jonas S. Friedenwald), li, 554
Blepharoptosis, Modified Motais for, (D. B. Kirby) lvii, 327; Operation for, (Robert G. Reese) liii, 26
Blind, Commission for, xliii, 420; The, in Germany, (Betty Hirsch) lvii, 178
Blind School Wassermann Tests in Missouri, (J. W. Charles and Harvey D. Lamb) xlvii, 455
Blindness, of Obscure Nasal Origin, (William Humes Roberts) liii, 19; from Gastro-Intestinal Hemorrhage, (G. H. Grout), xliii, 234, 268; in Missouri and St. Louis, (H. D. Lamb) lvi, 469; Report on British, (P. G. Doyne) lii, 478; A Standard for, xliv, 205
Blood Stream, Visibility of, with a Lens, (T. Harrison Butler) liii, 267
BLUE, R., Gram-Negative Diplococci in Conjunctival Sac, xliv, 652
BOIT, "Choroidal Detachment after Cataract and Glaucoma Operations," Notice of, xliii, 682
BOOKWALTER, C. F., Intranasal Dacryocystotomy, xlix, 568
BORDLEY, JAMES, JR., Malignant Uveitis and Thyroid Extract, xliv, 638; Ocular Manifestations of Disease of the Para-Nasal Sinus, l, 137; Papillœdema, liv, 158
BOTHMAN, LOUIS, and COHEN, SEYMOUR, Intraocular Pressure, lvi, 110
Brain, Frontal Lobe Tumor of, Autopsy, (Arnold Knapp) xlvii, 83; Hypophyseal Tumors of, and Visual Fields, (Clifford B. Walker and Harvey Cushing) xlvii, 119
BRAZEAU, Serous Tenonitis and Bilateral Papillœdema, lii, 355
BROCK, W., Idiopathic Iridocyclitis and Sympathetic Ophthalmia; xliv, 64
BROSE, L. D., Intraocular Foreign Body, li, 384; Ophthalmoplegia Interna from Lead Poisoning, xliv, 26
BROWN, ALBERT L., Rapid Dilation of Lacrimal Sac with Sea Tangle Probes, lvii, 397
BROWN, E. V. L., Anatomical Study of Conus in an Hyperopic Eye, xliii, 254; Localization of Streptococci in the Eye, xlv, 229; The Division of Ophthalmology in the University of Chicago School of Medicine, lvii, 190
BRUECKEN, A. J., Fibro-Adenoma of Ciliary Body, xliv, 490
BRUECKNER, A., "Grundzuege der Brillenlehre fuer Augenaertzte," liv, 109

BRUNER, ABRAM B., A Ring Sarcoma of Iris, liii, 162; The Visual Complex, liv, 372
BRUNS, H. DICKSON, Syphilis and Iritis, liv, 462
BURCH, F. E., Tendon Tucker and Suturing, li, 360, liv, 333
BURGE, W. E., Production of Cataract, xlvii, 12
BURGE, W. E., and NEILL, A. J., Lens Protection against Violet Light, xliv, 498
BURKE, J. W., Total Keratoplasty, l, 147
BURKY, EARL L., and WOODS, ALAN C., Lens Protein—New Methods for the Preparation of Beta Crystallin, lvii, 41; Lens Protein—The Isolation of a Third (Gamma) Crystallin, lvii, 464
BUTLER, T. HARRISON, "An Illustrated Guide to the Slit Lamp," lvii, 215; Dermoid of Cornea, lii, 469; Plastic Surgery of Lids in War, xlviii, 103; Retrobulbar Neuritis in a Baby, xlviii, 240; Ring Magnet, xlix, 247; Sarcoma of Lacrimal Sac, xliii, 16; Sclerotomy and Late Infection, xliv, 611; The Trephine in Chronic Glaucoma, l, 1; Tonometric Chart, xlix, 227; Visibility of Blood Stream with a Lens, liii, 267
BYERS, W. GORDON M., Encapsulated Angioma of Orbit, liii, 280; Histology of Eyes Lost after Trephining, xlvii, 462; Intermittent Exophthalmos, l, 569; Ophthalmology in Medical Curriculum, li, 566
BYRD, HIRAM, The Nasal Ganglion and Glaucoma, lvi, 162

CALHOUN, F. PHINIZY, Eye Complications from Typhoid Inoculations, xlviii, 553
CAMPBELL, DON M., CARTER, JOHN M., and DOUR, HOWARD P., X-Ray Studies of the Naso-Lacrimal Passages, li, 462
Capsulotomy, (A. E. Ewing) xlix, 520; With Subconjunctival Flap in Cataract Extraction, (H. W. Wandless) xliii, 494
Carcinoma, Metastasis of, into Endothelioma of Optic Nerve Sheath, (M. Cohen and W. J. MacNeal) l, 90, 128
CARR, ANDY M., Pupil Contraction and Tension, xlvii, 177; Tension and Tonsillectomy, xlvii, 46
Caruncle, Cyst and Cystic Tumors at, and Sebaceous Cysts, (John Green, Jr.) li, 145
Case Records, (Conrad Berens, Jr., and Gertrude E. Sturges) lii, 259
CASSELL, JAMES WILSON, Dr. Henry Holland's Eye Clinic at Shikarpur, India, lvii, 380
Cataract, A Conjunctival Apron in Extraction of, (Julius Wolff) lii, 554; A Second Hundred Successive Extractions of, in the Capsule after Preliminary Subluxation with Capsule Forceps, (Arnold Knapp) l, 426; Blocking of Facial Nerve in Extractions of, (R. E. Wright) lii, 166; Blocking Fifth and Seventh Nerves in Operations for, (J. Mathews Robinson) liii, 570; Choice of Operation, (Dorland Smith) lii, 25; Congenital, and Microphthalmus, (Henry Smith) l, 422; Corneal Suture in Extraction of, (E. C. Ellett) l, 341; Drawn Up Pupil after Extraction of, (Henry Smith) l, 423; Early Senile, Ptosis, and After-Cataracts, (Henry Smith) li, 40; Expression of, in Capsule, (Henry Smith) lv, 213; Findings in Coralliform and Congenital, (F. H. Verhoeff) xlvii, 558; Forceps for Intracapsular Extraction of, (F. H. Verhoeff) xlv, 479; Intracapsular Expression of, (A. S. and L. D. Green) li, 338; Intracapsular Extraction of, (L. Mills) xlvi, 344; Lens Proteins, their Chemical Changes in the Production of Senile, (I. S. Tassman) lvii, 361; Non-Operative Treatment of, (Zenas H. Ellis) lvii, 46; One Hundred Operations a Day, (H. T. Holland) liii, 155; One Hundred and Eighty-three Extractions, Twenty with Capsulotomy, (C. B. Meding) xlvi, 331; One Hundred Extractions of, in Capsule, (A. Knapp) xliv, 1; Operation for Shrunken, in Adolescents, (Arnold Knapp) lvii, 594; Prevention of Loss of Vitreous in Extraction of, (W. A. Fisher) xliv, 330; Production of, (W. E. Burge) xlvii, 12; Serum and Lens Antigen Extract

Cataract—(*Continued*)
for Prevention and Cure of, (A. Edward Davis) liv, 172; Smith's Intracapsular Extraction, (Clarence King) l, 440; Subconjunctival Sutures in Operations for, (F. H. Verhoeff) lvi, 137; The Capsule in Extraction of, xlix, 275; The Glaucoma of Hypermature, (H. Gifford) lvi, 457; The Iris in Operations for, (Henry Smith) lvi, 105; Treatment after Operations for, (J. W. Millette) l, 446; Treatment of, (Henry Smith) l, 514; Trial Lenses for Refraction after Extraction of, (D. N. Dennis) xliii, 128; Uncomplicated, (Derrick T. Vail) xlv, 307
Cataract Extraction Two Safety Measures in, (Walter S. Atkinson) lvii, 275; in Capsule (Arnold Knapp) xlvi, 27
Cataract Extractions, The Capsule in, (Arnold Knapp) l, 115
Cataract Operations, Backing Out of, (H. Gifford) l, 109; Observation, Management, and Treatment of the Patient Before and After, (Walter H. Snyder) l, 120
Cavernous Sinus Thrombosis, (Dorland Smith) xlvii, 482
CHAMBERS, STANLEY O., Literature of Treatment of Syphilitic Eye Diseases, lvii, 412
CHAMBERS, TALBOT R., Trifocal Minus Wafers, li, 154, 172
CHANCE, BURTON, Late Neuropathic Keratitis after Gasserian Ganglion Injections of Alcohol for Neuralgia, xlix, 621
CHARLES, J. W., Positive Tubercular Reaction in Perforating Sarcoma, xlviii, 568
CHARLES, J. W., and LAMB, HARVEY S., Wassermann Tests in Missouri Blind, xlvii, 455
CHENEY, ROBERT CARTWRIGHT, Orbital Abscess simulating Detachment of Retina: Anæmic Infarct of Optic Nerve, lii, 252
Chiasm and Optic Nerves, Primary Gliomas of, and of the Intracranial Portions of the, (Paul Martin and Harvey Cushing) lii, 209
Chloroma, with Ocular Lesions, (Martin Cohen) lvii, 238, 303
Choked Discs, Recession of, Following Operations for Brain Tumor, (Gilbert Horrax and Cameron Haight) lvii, 467
Chorioretinitis, Bilateral Circumscribed, with Detachment of Retina in Syphilis, (Arnold Knapp) l, 242; Globular Masses in Pupillary Margin, in Acute, (Harry and Jonas S. Friedenwald) lv, 113; Recurrent, from Various Causes, (C. E. Finlay) xlix, 430; Studies of the Visual Fields in Localized, (H. P. Wagener and John F. Gipner) lv, 470
Choroid, Angioma of, (J. M. Love) xliii, 607; Congenital Absence of, (Grimsdale) xlvi, 267; Flat Melanosarcoma of the, (James W. Smith) lvi, 156; Hemangioma of (S. B. Marlow) lvii, 484; Melanosarcoma of, in Brothers, (A. O. Pfingst and Stuart Graves) l, 431; Melanosarcoma of, with Phthisis and Iridocyclitis Bilateral, (Martin Cohen) liv, 58; Metastatic Carcinoma of, (J. A. Macmillan) li, 227; Mycosis Following Extraction, with Metastasis and Symptoms of Sympathetic Uveitis, (F. H. Verhoeff) liii, 517; Sarcoma of, (Edward Jackson and William C. Finnoff) xlvii, 72; Spontaneous Separation of the, (F. H. Verhoeff and J. H. Waite) lv, 38; Subretinal Exudate Simulating Tumor, Anatomical Examination, (Arnold Knapp) xlviii, 559
Choroiditis, from Alveolar Abscess, Vaccine Treatment, (E. H. Rowe) xlvi, 251
CHOU, C. H., A Typical Form of Familial Degeneration of the Cornea, (Fleischer) lvii, 574
Ciliary Body, Adenofibroma of, (A. J. Bruecken) xliv, 490; Tear of, (H. D. Lamb) lvi, 332
Ciliary Ganglion Anæsthesia, (H. S. Gradle) xliv, 270
CLAPP, C. A., Atypical Albuminuric Retinitis, xlix, 98; Weight and Percentage of Solids of Cataractous Lenses, xlv, 574
CLARK, C. F., Changes in Refraction in Forty Years, lvi, 52; Coloboma and Congenital Dislocation of Lens, xlviii, 475
CLARKE, ERNEST, Errors of Refraction and Accommodation, liv, 209

Clinical Research, Suggestions for, (Basil Graves) lv, 319
Cloquet's Canal Visible in the Living, with Hemorrhage into the Canal, (Otto Barkan) lvii, 502
COATS, G., Lymphoid Infiltration of Orbit, xliv, 221; Lymphoma and Lymphosarcoma of Conjunctiva, xliv, 235
Coats' Disease, Atypical, (L. W. Crigler) xlix, 287
COBB, PERCY W., Dark Adaptation, xlviii, 492
COHEN, MARTIN, A Mercury Tonometer, l, 326; Amaurotic Family Idiocy, lii, 140; Chloroma with Ocular Lesions, lvii, 238, 303; Distant Stereoscopic Vision, xlviii, 142, 168; Eyes in Polycythæmia, xlvii, 192, 203; Eye Symptoms in Fractured Skulls, xlvi, 258, 279; Intradural Tumor of Optic Nerve, xlviii, 19; Leukæmia and the Eyes, xlvii, 61; Lipæmia Retinalis with Hypotony in Diabetic Coma, l, 247; Melanosarcoma of Choroid, with Bilateral Phthisis Bulbi, and Iridocyclitis, liv, 58; Metastatic Ophthalmia after Pneumonia, xlv, 250; Orbital Neurinoma, liv, 426; Purpura Hemorrhagica, lii, 471; Retinal Hemorrhages in Bothriocephalus Anæmia, liii, 517
COHEN, MARTIN, and MACNEAL, WARD J., Metastasis of Carcinoma into Endothelioma of Optic Nerve Sheath, l, 128
COHEN, MARTIN, KILLIAN, JOHN A., and KAMNER, MILDRED, Comparative Chemical Studies of the Ocular Fluids, of Cerebrospinal Fluid and of the Blood, lvii, 59
COHEN, MARTIN, and MEEKER, LOUISE H., Multilocular Cyst of Optic Nerve Meninges, lvi, 211
COLLINS, E. TREACHER, "Arboreal Life and the Evolution of the Human Eye," lii, 512
COLLINS, TREACHER, and MAYOU, M. STEPHEN, "Pathology and Bacteriology of the Eye," liv, 617
Coloboma and Congenital Dislocation of Lens, (C. T. Clark) xlviii, 475
Color Sense, Examination of, (Helmbold) xlv, 270
Colorimeter, (Martin Cohen) lvii, 659
Conjugate Deviation with Astereognosis, (H. A. Riley) xlviii, 340
Conjunctiva, Amyloid Degeneration of, (R. E. Wright) liv, 280; Fusiform Bacilli in, and in Meibomian Glands, (S. R. Gifford) xlix, 477; Initial Development of, Papillary Bodies in the (Kazuo Hiwatari) xlix, 283; Keratoplasty, (Arnold Knapp) xlv, 359; Lymphoma of the, (G. Coats) xliv, 235; Melanosarcoma of, (Antonio S. Fernando) lii, 168; Melanosarcoma of the, (William Campbell Posey) lv, 131; Parinaud's Syndrome (?), (R. E. Wright) liv, 423; Streptothrix Infection of, (Arnold Knapp and James G. Dwyer) xlvii, 297; Tuberculosis of, (Felisa Nicolas) li, 379
Conjunctiva and Cornea, Molluscum Contagiosum in Diseases of, (Anton Elsching) li, 237
Conjunctivitis, Atrophying, (R. Kuemmell) xlvi, 147; Bacteriological Study of an Epidemic of, with Cell-Inclusion Conjunctivitis, (Hideyo Noguchi and Martin Cohen) xlv, 155; Purulent; in Infants, Incidence, Causes and Effect on Vision, (Anna W. Williams and Carolyn Rosenberg) xlv, 190
Conjunctivitis Tularense, (Derrick T. Vail) lv, 235
Conjunctivitis Tularense, (R. Sattler) xliv, 265
CONNELLAN, J. G., Intestinal Infections, xlviii, 350, 527
Conservation of Vision, (H. C. Greene and G. S. Derby) xliii, 262, 501
"Contributions to Ophthalmic Science," (E. Jackson) lv, 618
Conus, Study of a Temporal, in a Hyperopic Eye, (E. V. L. Brown) xliii, 254
Convergence Index as Measure of Convergence Power, (A. Duane) xliii, 476
Convergence Near Point, Determination of, and its Importance, (A. Duane) liii, 119
Copper Sulphate, (A. Duane) xliv, 481

Cornea, Accidental Splitting of, (A. C. Snell) xliii, 620; Atypical Epithelial Dystrophy, (William Campbell Posey) xlvi, 113; Blood Staining of the, (H. L. Begle) xliii, 393; Dermoid of, (T. Harrison Butler) lii, 469; Diplobacilli in Ulcers of, (H. W. Scarlett) xlvi, 219; Dystrophia of, (R. G. Reese) xliii, 57; Epithelial Dystrophy, Slit Lamp, (S. R. Gifford) liv, 217; Familial Degeneration of (Fleischer), (C. H. Chou) lvii, 574; Hæmosiderosis of the, in Diabetique Bronze, (B. Fleischer) xliii, 626; Keloid of, (H. D. Lamb) l, 535; Operation for Large Staphylomata of, (R. E. Wright) liv, 55; Optics of, (G. W. Vandegrift) xlix, 421; Radium Treatment of Tumor of, (A. F. Mattice) xliii, 237; Slit-Lamp in Study of Tubercles of, (B. T. and F. H. Haessler) li, 523; Surgery of Epithelioma of, (L. M. Francis) l, 331; Traumatic Surgery of Wounds of the, with Iris Prolapse, (P. Chalmers Jameson) lv, 465; Trephining of, in Glaucoma, (H. Tooke) li, 14

Corneal Epithelium, Dystrophic Opacity of the, (Nelson M. Black and F. H. Haessler) lvi, 151

Corneal Limbus Neoplasm; Mesothorium, (Koellner) xlvi, 130

Corneoscleral Margin, Histology of Superficial Layer of, (Kazuo Hiwatari) l, 10

Correspondence, on Extraction, lvi, 100; on Trachoma, lvi, 203; and on Miotics, lvi, 421

Couching, (G. Lindsay Johnson) liv, 466

COWAN, ALFRED, Cards for Visual Acuity, lvii, 283

CRIGLER, L. W., Atypical Coats', xlix, 287; Epibulbar Sarcoma, Macrosopically and Microscopically, xliv, 41; Pterygium Operation, li, 577

CRILE, GEORGE W., "Bipolar Theory of Living Processes," lv, 617

Critchett, Sir Anderson, In Memoriam, liv, 399

CROCKETT, R. L., Immuno-Therapy in Staphylococcic Infection of the Eye, xliii, 379

CROSS, G. H., Removal of a Lead Shot from the Eye, lvi, 564

CUPERUS, N. J., Mesothorium, xlvi, 126

CURDY, R. H., Anilin Injuries of the Eye, xlv, 243

CURRAN, E. J., Glaucoma Operation, Involving New Causation and Treatment of, lxix, 131; Subconjunctival Cauterization over Ciliary Body in Glaucoma, liv, 321

CUSHING, HARVEY, and BAILEY, PERCIVAL, Hemangioma of Cerebellum and Retina (Lindau's Disease), lvii, 447

CUTLER, COLMAN W., Disease of Optic Nerve and Relation to Posterior Nasal Sinuses, lii, 331

Cyclitis, Idiopathic, and Sympathetic Ophthalmia, (W. Brock) xliv, 64

Cyclophoria, Ames' Spectacle Device for Treatment of, (W. M. Lancaster) lvii, 322

Cyclopia, Teratogenesis, Embryology, and Cyclocephalic Monster, (Emory Hill) xlix, 597, l, 52

Cyclopic Eye, (Sh. Funaishi) lvi, 328

Cyst, Dermoid, (Harry S. Gradle and J. N. Stein) liii, 254

Cystotome Manœuvre in Capsular Extraction, (J. A. Kearney) xlviii, 62

Cystotomy, Peripupillary, (Falchi) xlvi, 135

Dacryoadenitis, Acute Infectious, (M. L. Foster) liv, 430

Dacryocystitis, Simulated, (John Green, Jr.) liv, 68

Dacryocystorhinoscopy, (A. Wiener) xlvi, 227

Dacryocystotomy, Intranasal, (C. F. Bookwalter) xlix, 568

Dacryops, (Otto Landmann) liv, 592

DARIER, A., "Traité Complete de Thérapeutique Oculaire," l, 598

Dark Adaptation, (P. W. Cobb) xlviii, 492

D'AUTREVAUX, "Histo-Bacteriologique Oculaire," lvj, 313

DAVIS, A. EDWARD, Serum and Lens-Antigen Treatment for Cataract, liv, 172

DAVIS, M. M., and HARTSHORN, E., Self-supporting Eye Clinic, xliii, 643
DAVIS, W. T., Vascular Lesions of Eye Ground, lvi, 477
DE BLASCOVICS, L., Ptosis Operation, Shortening of Levator and Tarsus, lii, 563; Traumatic Coloboma of Lids, xlvii, 404; Turning of the Tarsus, liv, 38; Unit of Visual Acuity and Its Practical Value, liii, 476
DE LIEBERMANN, L., and WERFELMAN, W. J., Thread Fixation of Lid in Tarsus Operations, lv, 462
Dengue, Ocular Symptomatology in, (William D. Gill) lvii, 269
DENNIS, D. N., Light Fields Apparatus, xliii, 624; Trial Lenses for Cataract Cases, xliii, 128
DERBY, GEORGE S., Brawny Scleritis, xlv, 20; Eye Symptoms after Exposure to Gases, xlix, 119; Eye Work with the British in France, xlviii, 217; Nature of So-called Koeppe Nodules, lviii, 561; Posterior Transillumination of the Eye, liii, 152; Social Service and Follow up Work, xlix, 495
DERBY, G. S., and CARVELL, MAUD, Anterior Ocular Tuberculosis, lvi, 523
DERBY, G. S., and GREENE, H. C., Conservation of Vision, xliv, 186, 438
DERBY, G. S., and VERHOEFF, F. H., Sarcoid of Eyelid, xlvi, 342
DERBY, G. S., WAITE, J. HERBERT, and KIRK, E. B., Light Sense in Early Glaucoma, lv, 575
DERBY, DR. HASKET, Obituary of, xliii, 687
Dermoid Cysts, Trichloracetic Acid for, (H. Gifford) lii, 448
Descemet's Membrane, Excrescences of, (Daniel B. Kirby) liv, 588, 598
DE SCHWEINITZ, G. E., Ocular Symptoms in Hypophyseal Disease with Acquired Syphilis, l, 203, 268; "Diseases of the Eye," xlvi, 608, li, 301, liv, 210; Eye Phenomena in Psychoneuroses of War, xlviii, 419; Progressive Atrophy of Iris, lvi, 10
DE SCHWEINITZ, G. E., and HOW, H. W., Pituitary Body Disease and Glandular Treatment, xlvi, 19
DE SCHWEINITZ, G. E., and WOODS, ALAN, Ocular Symptoms, Particularly Fundus Pictures in Erythræmia, lv, 21
DEWEY, KAETHE W., Eye Diseases from Teeth in Relation to Lymphatic System, xlix, 367
Diabetes, Ocular Nerves in, (M. Hoffmann) xliii, 39
Diet, in Prevention of Post-Operative Infections, (G. H. Bell) lii, 198
DIMITRY, U. J., Eviscero-Neurotomy, with Endothesis a Substitute for Enucleation, li, 365
Diplococci, Gram Negative, in Conjunctival Sac, (R. Blue) xliv, 652
Dispituitarism, with Contracted Fields Relieved by Gland Therapy, (W. Timme) xlvi, 151
Distance Judgment with Semaphores and Screens, (H. J. Howard) xlviii, 461
Distance Stereoscope, (M. Cohen) xlviii, 142; (H. J. Howard) xlviii, 168
Divergence, Excess, Diagnosis and Treatment, (John H. Dunnington), lvi, 344
Divergence Paralysis in Epidemic Encephalitis, (J. H. Dunnington) lii, 39
DOLMAN, PERCIVAL, The Maddox Rod and Its Defects, xlix, 194
DOLMAN, PERE, Maddox Rod, xlviii, 503
DONNELL, N. R., Perithelioma of Upper Lid, liii, 411; Strong Silver Nitrate for Trachoma, liii, 433
DOUBLER, F. H., and MARLOW, S. B., Hemorrhage into Optic Nerve Sheath from Cerebral Artery Aneurism, xlvi, 533
DOYNE, P. G., British Blindness and Its Prevention, lii, 478
DUANE, ALEXANDER, Anomalies of Accommodation, xlv, 124; Basic Diagnostic Principle in Motor Anomalies, xlviii, 1; Convergence Index and Converging Power, xliii, 476; Convergence Near Point, Determination and Importance, liii, 119; Obituaries of, lvi, 66; Post-Graduate Course in Ophthalmology, xlix, 17; Practical Points in Refraction, xlix, 349; Practical Points in Eye Work, xliv, 481; Projection and Double Vision, liv, 233; Recur-

DUANE, ALEXANDER—(*Continued*) rent Paralyses of Eye Muscles and Ophthalmoplegic Migraine, lii, 417, 436; Subnormal Accommodation, liv, 566; Tangent Curtain, xliii, 591; Torticollis and Tenotomy of Inferior Oblique, xlv, 33

DUKE-ELDER, W. STEWART, Intraocular Fluids, lvii, 100; Recent Advances in Ophthalmology, lvii, 210

DUNN, JOHN, Automatic Reflex and Argyll-Robertson Pupil, xlvi, 193; Exophthalmic Goiter, xlvii, 248; Eye Strain Headache, xlvii, 147; Pathological Pupil Cases and Argyll-Robertson Pupil, xlvii, 469; Pupil in Glaucoma, xlviii, 126; Pupillary Symptoms in Embolus of Central Retinal Artery, with Reference Especially to the Primary Reflex, xlix, 191; Surgery of Meibomian Cysts, xlviii, 43; Tonsil Infection in Iritis and Choroiditis, xlvi, 446; Vestibular Nerve Problem, xlvii, 354

DUNNINGTON, JOHN H., Divergence Excess, Diagnosis and Treatment of, lvi, 344; Divergence Paralysis in Epidemic Encephalitis, lii, 39

DUNPHY, EDWARD BLAKESLEY, Ophthalmoplegia Internuclearis, lv, 54

DWYER, J. G., Intestinal Eye Infections, xlvii, 261, 305

EAGLETON, WELLS P., "Brain Abscess, Its Surgical Pathology and Operative Technique," lii, 308; "Cavernous Sinus Thrombosis," lvi, 311

Ear and Eye Relations and the Barany Tests, (H. M. Langdon and I. H. Jones) xlvii, 348

Echinococcus, Subconjunctival, (George R. Hare) lv, 357

EDMUND, CARSTEN, and MOELLER, H. ULRIK, Vision in Light of Reduced Intensity, liv, 531

ELLER, J., The Significance of the Ciliary Epithelium in the Absorption of Vitreous Hemorrhages, lvii, 134

ELLETT, E. C., Corneal Suture in Extractions, l, 341; Heterochromia Iridis, Heterochromic Cyclitics, etc., xlvi, 562

ELLIOT, R. H., "Indian Couching Operation," xlvii, 211; "Sclero-Corneal Trephining in Glaucoma," xliii, 467; "A Treatise on Glaucoma," lii, 310; The Filtering Scar, li, 433; "Tropical Ophthalmology," l, 303

ELLIS, ZENAS H., Non-Operative Treatment of Cataract; Lens Antigen Treatment, lvii, 46

ELSBERG, C. A., Papillœdema in Brain Tumors and Its Surgical Treatment, liii, 307, 384

ELSBERG, C. A., and KRUG, E. F., Hypopituitarism; Glandular Treatment, xlvi, 97

ELSCHNIG, ANTON, Molluscum Contagiosum as a Cause of Conjunctival and Corneal Diseases, li, 237; Operation for Retinal Detachment, xliv, 163

Encephalitis, Blindness During, liv, 165; Lethargica, (H. Woods) xlviii, 536; Ocular Manifestations of, (Ward A. Holden) l, 101

Endocrines in Women, Eye Diseases from, (O. R. Lourie) liv, 469

Endophthalmitis from Retained Foreign Body Phagocyst, (Derrick T. Vail and F. H. Verhoeff) lvi, 5

Endophthalmitis (Phacoanaphylactic?) (A. Roetth) lv, 103, 610; Phacoanaphylactic, (F. H. Verhoeff) l, 296

Enophthalmos, Congenital, (A. Lutz) xlvi, 243

Entropion, Metal Sutures for Spastic, (Ralph L. Lloyd) liii, 566

Enucleation, (F. Fergus) xliii, 618; with Implantation of Gold or Glass Sphere, (A. Greenwood) xliii, 1

Ephedrine, Experiences with, in Ophthalmic Practice, (Mark J. Schoenberg) lvii, 272

Epibulbar Neoplasms, Non-Surgical Treatment of, (Edward B. Heckel) li, 141

Epiphora, (A. Wiener) xlviii, 334

Episcleritis, Diet Rich in Meat Proteins, (Sinskey, H. L., Levin, M. B., and Sacks, B.) l, 526

Erythæmia, Fundus and Ocular Symptoms in, (G. E. de Schweinitz and Alan Woods) lv, 21

EVANS, JOHN N., Synchysis Scintillans in Anterior Chamber, liii, 142

Evisceration, (H. Gifford) xliv, 162
Eviscero-Neurotomy with Endothesis in Place of Enucleation, (T. J. Dimitry) li, 365
EWING, A. E., Capsulotomy, xlix, 522; Duct Conservation in Lacrimal Abscess, liii, 31
Exenteration and Enucleation, Novocain Anæsthesia in, (H. Illing) xlv, 46
Exenteration vs. Enucleation, (H. S. Gradle) xliv, 29, 154, 270
Exophthalmic Goiter, Surgery of, Corneal Suppuration in, (Arnold Knapp) xlvii, 173
Exophthalmometer, (W. Lohman) xlv, 275
Exophthalmos, Intermittent, (W. Gordon M. Byers) l, 569; Traumatic Pulsating, (A. J. Bedell) xliv, 139
Eye, Autogenous Vaccines in Diseases of, (L. Herbert Lanier) li, 349; Deficiency Diseases of, (Francis Heed Adler) lvi, 592; Diseases of the, and Adnexa, Due to Fungi and Higher Bacteria, (S. R. Gifford) lvii, 224; Dislocation of, Without Orbital Fracture or Scleral Rupture, (S. P. Oast) liv, 191; Function in the Structure of the, (E. Uhlenhuth) xlvii, 401; Immuno-Therapy in Staphylococcic Infection of the, (R. L. Crockett) xliii, 379; Manifestations of Internal Secretions in the, (E. Fuchs) li, 307; Metal in the, After Magnet Extraction, (A. F. Mattice) xliii, 20; Nutrition of, (Andrew Rados) lvi, 567; Penetrating Injuries of, lv, 172; Posterior Transillumination of, (G. S. Derby) liii, 152; Protein Injections in Diseases of the, (B. W. Key) li, 471; Sarcoma of, (L. W. Crigler) xliv, 229; The, and the Endocrines, (Percy Fridenberg) xlix, 485; Two Early American Works on the, (George H. Jackson) lvii, 265
Eye and Lids, Epithelioma of, (Dunbar Roy) xlviii, 563
Eye and Teeth, (W. F. C. Steinbugler) xlvii, 182
Eye Clinic, Report on a Self-Supporting, (M. M. Davis and E. Hartshorn) xliii, 643
Eye Disease, Chronic Gonorrhœal Prostatitis as a Possible Cause of, (A. H. Thomasson) lii, 546
Eye Diseases from Teeth and Lymphatic System, (Kaethe W. Dewey) xlix, 367
Eye, Ear, and Vestibular Organs, Relations Between, (J. Van der Hoeve) li, 321
Eye Function, Neurotic Disturbance of the, (Lawrence K. Lunt and Austen Fox Riggs) lii, 313
Eyelid, Chancre of, (H. H. Tyson) xliv, 410
Eyelids, Allergic Reaction of, (Eugene M. Blake) liii, 272; Autogenous Grafts for Restoration of the, (E. B. Heckel) xlviii, 311; Collodion Closure after Infiltration Anæsthesia, (William D. Gill) lvii, 269; Plastic of, from War Injuries, xlix, 35; Plastic Surgery of, (T. Butler Harrison) xlviii, 103; Syphilitic Gumma of, (E. B. Heckel and L. G. Bernheimer) liv, 352
Eye Specimens, Preservation of, (E. Greef) xliv, 182
Eye Strain Headache, (John Dunn) xlvii, 146
Eye Strain, Relation of, to Functional Neurosis, (F. W. Marlow) lvii, 339
Eye Surgery in War, (G. H. Grout) xlviii, 227
Eye Surgery, Lamp for, (A. Maitland Ramsay) li, 152
Eye Work, Practical Points in, (A. Duane) xliv, 481

FALCHI, Peripupillary Cystotomy, xlvi, 135
Fat-Transplantation after Enucleation, (A. N. Alling) xlviii, 263
FEIGENBAUM, Palestine Campaign against Trachoma, lv, 453; Trachoma in Palestine, lv, 159
FEINGOLD, MARCUS, Progressive Muscular Degeneration, Familial, xlv, 533
FERGUS, F., Easy Enucleation of the Eye, xliii, 618; Salvarsan and Other Eye Remedies, xlvii, 29
FERNANDO, ANTONIO S., Eye Diseases in Philippine General Hospital, lii, 170; Causes of Blindness Amongst Filipinos, li, 374; Melanosarcoma of Conjunctiva, lii, 168

FERRER, C. E., and RAND, G., Apparatus for Testing Light- and Color-sense, and Color Mixing, lv, 453
Fibrolysin, (R. S. Lamb) xlvi, 233
Field-Taking, Tangent Screen for, (A. H. Thomasson) lv, 545
FIELDS, S. O., Intermittent Ophthalmomalacia, li, 233
Filipinos, Causes of Blindness Amongst the, (Antonio S. Fernando) li, 374
Filtration Scar, (R. H. Elliot) li, 433, 499
FINLAY, C. E., Bitemporal Contraction of Visual Fields in Pregnancy, lii, 50; Eyelid Chancre, xliv, 410; Recurrent Chorioretinitis Due to Different Causes, xlix, 430
FINNOFF, WILLIAM C., Ocular Tuberculosis, Experimental and Clinical, liii, 130; The Ciliary Glands, xliv, 626
FISHER, W. A., "Ophthalmoscopy, Retinoscopy and Refraction," lii, 414; Prevention of Loss of Vitreous in Operations, xliv, 626; "Senile Cataract, Methods of Operating," lii, 621
Fistula, Closure of Traumatic Subconjunctival and Corneo-Scleral, (H. S. Gradle) l, 154
FLEISCHER, BRUNO, Keratoconus with Hæmosiderosis; Hæmosiderosis in Diabetes, xliii, 626
FLURY, Scientific Treatment of Arteriosclerosis with Iodide of Potash, xliii, 405
Foreign Body, Intraocular, (L. D. Brose) li, 384
FORONI, CARLO, Keratectomy, xlvi, 57
FOSTER, MATTHIAS LANCKTON, Locally Infected Dacryoadenitis, liv, 430
FOX, L. WEBSTER, "A Practical Treatise on Ophthalmology," xlix, 548; Trachoma Amongst Indians, liii, 166
FRANCIS, LEE MASTEN, Removal of Magnetic Bodies from Vitreous, xlix, 198; Surgical Treatment of Epithelioma of Cornea, l, 331, 369
FRANKE, E., "Die Augenaertzliche Praxis," liii, 632; "Ocular Therapeutics," liv, 619
FRAZIER, CHARLES H., Control of Pituitary Lesions Affecting Vision by Combined Surgical, X-Ray, Radium Treatment, l, 217
FRIDENBERG, PERCY, The Eye and the Endocrine System, xlix, 485
FRIEDENWALD, HARRY, Vascular Lesions of Retina in Young People, liv, 111
FRIEDENWALD, HARRY, and JONAS S., Globular Masses on the Pupillary Margin in Circumscribed Chorioretinitis, lv, 113; Melanosis of Lids, Conjunctiva, and Sclera, with Warts on Iris, liv, 51
FRIEDENWALD, JONAS S., Blepharochalasis, lii, 367; Coloboma of Iris, liv, 349; Virus of Herpes Zoster, lii, 103
Frontal Bone Fracture, (Arnold Knapp) xlvi, 272
FUCHS, ADALBERT, "Atlas of the Pathology of the Eye," lvii, 106; "Atlas der Histopathologie des Auges," liii, 628
FUCHS, E., Ocular Manifestations of Internal Secretions, li, 307; Presbyopia, li, 21, 63; "Text Book," xlvi, 609; "Text Book of Ophthalmology," lii, 308
FUMIO, YANO, Unusual Pterygium, l, 239
FUNAISHI, SH., Cyclopic Eye, lvi, 328
Fundus, One Hundred Examinations of the, as an Index to Arterial Disease, (S. A. Agatston) lvii, 386
Fundus Oculi, Vascular Lesion of, (W. T. Davis) lvi, 477
Fusion Impulse, Therapeutic Suppression of, (Ellice M. Alger) lv, 438

GABRIELEDEA, A., "Des Conjonctivites," li, 200
GABRIELS, J. A. C., Percentage Vision, and Snellen Notation, lv, 58; Snellen Symbol and Visual Values, lv, 447
GAGE, MARGARET, Myopia in Massachusetts Infirmary, xlvi, 154
GAMBLE, WILLIAM E., Weak Atropine Solutions in Asthenopia, lii, 160
Gases, Eye Manifestations from Exposure to Poisonous, (G. S. Derby) xlix, 119
Giant Magnet, Correct Use of, (Otto Haab) xlv, 256

GIFFORD, HAROLD, Backing out of Cataract Operations, l, 109; Evisceration, xliv, 162; Glaucoma, liv, 337; Glaucoma of Hypermature Cataract, lvi, 457; Trichloracetic Acid for Dermoid and Other Cysts, lii, 448
GIFFORD, HAROLD and SANFORD R., Molluscum Conjunctivitis, l, 227
GIFFORD, SANFORD R., Blood Count in Sympathetic Ophthalmia, xlvii, 327; Diseases of the Eye and Adnexa from Fungi and Higher Bacteria, lvii, 224; Epithelial Dystrophy of Cornea with Slit Lamp, liv, 217; Fatty Infiltration of Anterior Segment (Xanthomatosis bulbi), lvi, 281; Fusiform Bacilli on Conjunctiva and in Meibomian Glands, xlix, 477; Ocular Sporotrichosis, li, 540, liii, 264; Pathology of Uveitis, li, 483; Radium in Leucosarcoma of Iris, xlvii, 241; Some Modern Preparations for the Treatment of Glaucoma, lvii, 612
GILBERT, W., Brawny Scleritis, xliv, 545
GILL, WILLIAM D., Collodion to Keep the Eyes Closed after Infiltration Anæsthesia, lvii, 259; Ocular Symptomatology in Dengue, lvii, 628
GILLIES, H. B., "Plastic Surgery of the Face," l, 299
Glass Ball Implantation, (H. J. Howard) xlviii, 263
Glaucoma, A Symposium, (W. O. Nance and W. H. Peck) xlv, 104; Acidosis and Œdema Related to (Henry Smith) xlv, 1; Conception of Visual Field Changes in Chronic, (Luther C. Peter) lvi, 337; Cyclectomy for, (F. H. Verhoeff) liii, 228; Endocrine Origin of, (Joseph Imre) liii, 205; from Disturbances of Intraocular Fluid Exchange, (J. Herbert Waite) lvi, 460, 522; Glaucosan, Glaucosan Drops, and Amine-Glaucosan Drops, (Carl Hamburger) lv, 533; in Generalized Vascular Nævus of the Skin, Case with Angiomatous Changes in the Iris, (Arnold Knapp) lvii, 219; Inherited Adrenalin Reaction (Knapp), (M. J. Schoenberg) liv, 282; Iris Prolapse Technique, (H. Herbert) li, 203; Its Cause, Diagnosis and Miotic Control, (Wm. Campbell Posey) xlix, 293; Light Sense in Early, (G. S. Derby, Herbert Waite, and E. B. Kirk) lv, 575; Medical Side of, (Arnold Knapp) xlv, 544; Modern Preparations Used in the Treatment of, (S. R. Gifford) lvii, 612; Modified Elliot-Lagrange, (Derrick T. Vail) lii, 346; New Technique, (E. Török) lii, 574; Non-Operative Treatment of Chronic, (Searle B. Marlow) lvii, 165; One Hundred Cases of Primary, at Moorfields, Visual Results, (Charles Maghy) xlix, 390; Operation Involving New Causation and Treatment of Chronic Primary, (E. J. Curran) xlix, 131; Operative Treatment of, (John E. Weeks) xlix, 316; Pathogenesis of, (F. H. Verhoeff) liv, 20; Practical Points in Operative Treatment of, (G. Lindsay Johnson) xliii, 7; Pupil in, (John Dunn) xlviii, 126; Radium for, (Schoenberg) xlviii, 485, 522; Subconjunctival Cauterization of Sclera, to Reduce Tension in, (E. J. Curran) liv, 321; Sclerocorneal Trephining in, (W. H. Wilmer) xlv, 333; Scotoma of, (A. H. Thomasson) lvi, 319, 381; Scotometry in Diagnosis and Treatment of, (A. H. Thomasson) lvii, 160; Spontaneous Hypotony in Juvenile, (Alfred H. Riedel) l, 457; Technique of Trephining for, (Frederick Tooke) li, 14; Temporary and Permanent Disturbances of Dark-Adaptation in, lvii, 550; Tonometry of, (William McLean) xlix, 301; Trephine in Chronic, l, 1, (T. Harrison Butler) l, 1; Trephining for, (W. R. Parker) xlvi, 1; Trephining of Cornea in, (L. Tooke) li, 14; Visual Field Examination, (W. Lohlein) xlv, 51; Visual Fields in, (Luther C. Peter) xlix, 309; with Herpes Zoster, (J. E. Weeks) xlvi, 68, 460; Without Tension, (H. S. Gradle) xlvi, 117; X-Ray Treatment of, (Ralph L. Lloyd) lvi, 445
Glaucoma and Meckel's Ganglion, (M. H. Post, Jr.) l, 317
Glaucoma and Nasal Ganglion, (Hiram Byrd) lvi, 162

Glioma, Bilateral, (Arnold Knapp) xlvi, 272; Unsuccessful Use of Radium in Bilateral, (Arnold Knapp) xlix, 575
Glioma and Atrophy of Eye, (D. F. O'Connor) xlvi, 298
Glioma and Pseudo Glioma, Slit-Lamp Diagnosis, (T. Harrison Butler) lv, 155
Goalwin, Clinical Value of X-Ray Pictures of Optic Canal, lv, 1
Goiter, Causes of Exophthalmic, (John Dunn) xlvii, 248; Double Luxatien of Eyes in Exophthalmic, (W. R. Parker) li, 1
GOLDENBURG, Anomalous Nerve Heads, xliv, 246
GOLDSTEIN, ISADORE, Embryology of Tenon's Capsule, lii, 327
Gonorrhœa Endogenous Affections of Cornea from, (F. Pincus) xliii, 136
GOUGELMANN, PIERRE, Artificial Eyes after Substitutes for Enucleation, xlviii, 268
Gout and Diseases of the Eye, (L. Litchwitz) xliii, 25
GRADLE, HARRY S., Cavernous Lymphangioma of Orbit, xlix, 520; Closure of Traumatic Subconjunctival, Sclerocorneal Fistula, l, 154; Glaucoma without Perceptible Tension, xlvi, 117; Removal of Eyeball, xliv, 29, 154, 270; Slit Lamp, li, 264; Subjective Examination of Pupillary Reflexes, xliii, 377; Successfully Operated Sellar Cyst, lv, 431; Unilateral Congenital Atresia of Puncta, l, 349
GRADLE, HARRY S., and STEIN, JULIUS C., Dermoid Cyst, liii, 254
GRAVES, BASIL, Slit-Lamp Study, lv, 319
GREEF, D., Cat-like Pupils in Man, xliii, 146
GREEF, R., "Guide to Microscopic Examination of the Eye," xliii, 233, 467; Preservation of Eye Specimens, xliv, 182
GREEN, A. S., and L. D., Intracapsular Expression-Extraction of Cataract, li, 338
GREEN, F. W. EDRIDGE, "The Physiology of Vision, with Special Reference to Color Vision," xlix, 550
GREEN, JOHN, JR., Cyst and Cystic Tumors of Caruncle and Sebaceous Cysts, li, 145; Palpebral and Conjunctival Affections Associated with Refractive and Muscular Errors, l, 158; Radium and Operation in Basal Cell Carcinoma of Orbit, lii, 75; Swelling Simulating Dacryocystitis, liv, 68; Trephining in Detachment, xlvi, 104
GREENE, H. C., Conservation of Vision, xliv, 186, 438
GREENE, H. C., and DERBY, G. S., Conservation of Vision, xliii, 262, 501
GREENWOOD, ALLEN, Enucleation, with Gold or Glass Implantation, xliii, 1; Lacrimal Sac Extirpation, l, 234
GREENWOOD, ALLEN, DE SCHWEINITZ, G. E., and PARKER, WALTER, "Military Ophthalmic Surgery," xlvii, 437
GRISCOM, J. MILTON, and MONROE, MARGARET M., Visual Field Studies in Optic Neuritis in Sinus Diseases, lvi, 244
GROTHUYSEN, G., Cyclical Third Nerve Paralysis, xliv, 666
GROUT, E., Permanent Impairment of Vision after Gastro-Intestinal Hemorrhage, xliii, 234, 269
GROUT, GERALD H., War Eye Surgery, xlviii, 227
GRUENING, H., Death of, xliii, 368, 587
GWATHMEY, JAMES T., General Anæsthesia in Eye Practice, liii, 579

HAAB, OTTO, Correct Use of Giant Magnet for Eye Operations, xlv, 256
HAESSLER, BERTHA B., and F. HERBERT, Slit-Lamp Microscopy for Development of Corneal Tubercles, li, 523
HAESSLER, F. HERBERT, Heterochromia Iridis, liv, 298
HAJEK, M., "Physiology and Treatment of Accessory Cavities Inflammations," lv, 616
Hallucinations, Optic, (O. Schirmer) xlvi, 247
HAMBURGER, CARL, Glaucosan, Glaucosan Drops, and Amine-Glaucosan Drops in Glaucoma, lv, 533

HANKE, VIKTOR, "Das Auge; Seine Schaedigungen, etc.," lvi, 520
HANSELL, HOWARD F., Eye Diseases and the Tonsils, xlvii, 572
HARDY, W. F., Retinal Lipæmia, l, 543
HARE, GEORGE R., Subconjunctival Ecchinococcus, lv, 367
HARROWER, DAVID, Iridotasis, xlvii, 37
HARTRIDGE, G., "The Ophthalmoscope," xlix, 548
HAZEN, E. H., Ocular Orientation, Dependent on the Muscular System, xlix, 434
HECHT, SELIG, Visual Acuity and Illumination, lvii, 564
HECKEL, EDWARD B., Autogenous Grafts in Restoration of Eyelids, xlviii, 311; Diphtheria Antitoxin in Sympathetic Ophthalmia, lvii, 54; Non-Surgical Treatment of Malignant Epibulbar Neoplasms, li, 141; Radium in Melanosarcoma of Orbit, xlv, 465
HECKEL, EDWIN B., and BEINHAUER, LAWRENCE G., Syphilitic Gumma of Eyelid, liv, 352
HEINE, L., "Die Krankheiten des Auges in Zusammenhang mit der Inneren Medizin," li, 614
HELMBOLD, Color Sense Examination, xlv, 270
HELMHOLTZ, "Physiological Optics," liv, 211, lv, 316; "An Ophthalmoscope," xlvi, 610
Hemianopsia, Bitemporal, Instrument for Accurate Measurements of, (C. B. Walker) xliv, 369
HEPBURN, MALCOLM L., "The Ophthalmology of General Practice," lii, 413
HERBERT, H., Iris Prolapse, Technique in Glaucoma Operations, li, 203; "The Operative Treatment of Glaucoma," lii, 509
Herpes Along Fifth Nerve after Blue Stone Application in Conjunctivitis, xlix, 628
Herpes Frontalis and Faulty Oculo-Muscular Adjustment, (Charles W. Stevens) liii, 60, 74
Herpes Zoster, Neuritis and Glaucoma Following, xlviii, 364; Virus of, (Jonas S. Friedenwald) lii, 103
HERRENSCHWAND, F. V., "Pathogenische Microorganismen des Auges." lvi, 515
HERTEL, E. L., "Behandlung der Tuberkulose Augenkrankheiten," xliii, 677
HERTEL, PROF. B., "Stilling's Tafeln zur Pruefung des Farbensinn," lvi, 312
HESS, CARL, Obituary, lii, 624
Heterochromia, Ætiology of, (J. Bistis) xliv, 433
Heterophoria, Treatment of, in Ametropia, (Herbert Wright Wooton) liv, 440
HEYL, T., After-Cataracts, and Immature Extractions, xlvi, 53
HILL, EMORY, Bilateral Detachment in Two Successive Pregnancies, liii, 137; Cyclopia, Teratogenesis, Embryology and a Cyclocephalic Monster, xlix, 597, l, 52
HINSHELWOOD, JAMES, "Congenital Word Blindness," xlvii, 213
HIRSCH, BETTY, The Adult Blind in Germany, lvii, 178, 206
HIRSCHBERG, J., "England's Augehaertzte," xliv, 604
HIRSCHBERG, JULIUS, In Memoriam, liv, 418
HIWATARI, KAZUO, Development of Papillary Bodies in the Conjunctiva, xlix, 283; Histology of the Superficial Layer of the Corneo-Scleral Region, l, 10; Trachoma and Normal Histology of the Conjunctiva, xlix, 82
HOFFMAN, MICHAEL, Ocular Nerve Diseases in Diabetes, xliii, 39
HOLDEN, W. A., Acute Disseminated Myelitis with Retrobulbar Optic Neuritis, xliii, 231; Ocular Manifestations of Epidemic Encephalitis, l, 101; Ocular Manifestations of Multiple Sclerosis, li, 114; Papilloedema, liii, 316, 384; "The Eye in Nervous Diseases," xlvii, 221
HOLLAND, H. T., One Hundred Extractions in a Day, liii, 155
HOLLANDER, A. R., and COTTLE, M. H., "Physical Therapy for Eye, Ear, Nose, and Throat," lv, 616
HOLLOWAY, T. B., Asteroid Hyalitis, xlvii, 50
HOLMES, CHRISTIAN R., Extirpation of Lacrimal Gland for Epiphora, xlviii, 323, 374
HOPKINS, J. C., Syphilitic Lesions of the Eye, lvi, 500; Treatment of, lvi, 543

HORRAX, GILBERT, Papillœdema in Neurology, liv, 130
HORRAX, GILBERT, and HAIGHT, CAMERON, Recession of Choked Disc After Operation for Brain Tumor, lvii, 467
HOWARD, HARVEY J., Apparatus for Testing Accommodation, xlix, 182; Glass Ball Implantation, xlviii, 265; Judgment of Distances with Semaphore and Screen, xlviii, 462; Tenotomy of Inferior Oblique, xlix, 381
HURST, ARTHUR F., "Psychology of the Special Senses and Their Functional Disorders," l, 599
Hyalitis, Asteroid (T. B. Holloway) xlvii, 50
Hydrophthalmus, (Eugene M. Blake) liv, 1
Hypertension, Fundus Changes in, with or without Nephritis, (Aaron Barlow) lvi, 356
Hypophyseal Disease, Eye Symptoms in Subjects of, with Acquired Syphilis, (G. E. De Schweinitz) l, 203
Hypophysis, Radium and X-Rays in Tumors of the, (Douglas Quick) xlix, 256
Hypopituitarism, Cured by Glandular Treatment, (C. A. Elsberg and E. F. Krug) xlvi, 97
Hypopyon Ulcer, lvi, 493
Hysteria, Blindness as a Symptom, (T. H. Ames) xliii, 357

IGERSHEIMER, "Das Shicksal von Patienten mit Keratitis Parenchymatose, etc.," xliii, 684
ILLING, H., Local Novocain Anæsthesia in Exenteration and Enucleation, xlv, 46
Illusions, Ocular, (M. Lindsay Johnson) lvi, 465
Immature Cataracts, Problem of the, (Homer E. Smith) l, 365
Immunology in Ophthalmology, (Alan C. Woods) liii, 321
IMRE, JOSEPH, Protection Lenses, lvi, 111; Endocrine Origin of Glaucoma, liii, 205
India, Eye Clinic of Dr. Holland at Shikarpur, (James Wilson Cassell) lvii, 380
Indians, Trachoma Amongst, (L. Webster Fox) liii, 166
Inferior Oblique, Isolated Paralysis of, (Searle B. Marlow) lii, 12; Tenotomy of, (Wm. Campbell Posey) xlv, 137, (H. J. Howard) xlix, 381
Injuries of Eye, Compensations for, (Albert C. Snell) liii, 36, 172
Internal Carotid, Effect of Aneurysm of, on Vision, (J. R. Shannon) xlvi, 518
Intestinal Eye Infections, (J. J. Conellan), xlviii, 350
Intestinal Tract and Eye Diseases, (J. G. Dwyer) xlvii, 261, 305, xlviii, 344, 527
Intracapsular Extraction, (G. L. Johnson) li, 548
Intracranial Pressure, Signs in Eye, (W. Sharpe) xlvi, 320, 363
Intraocular Pressure and Tonometry, (W. McLean) xlviii, 23
Iridocyclitis, Atropine and Epinephrin in, (Frank H. Rodin) lvi, 277; Discussion on, lvii, 533; Intradermal Test with Uveal Pigment in Traumatic, (Arnold Knapp) liv, 252; Sympathica Following an Iridectomy for Secondary Glaucoma, (C. W. Tooker and H. D. Lamb) liii, 439; Syphilitica and Its Causes, (E. L. Zimmermann) liii, 558; Treatment of, with Foreign Proteids, (F. H. Verhoeff) l, 193; Tubercular with Fatty Degeneration and Infiltration, (Otto Landsberg) liv, 261; Tuberculous, Treatment of, with Slit-Lamp and Tuberculin, (Clarence King) lv, 563
Iridocyclitis and Anaphylaxis (Alan C. Woods) xlvii, 161
Iridotasis, (David Harrower) xlvii, 37; Histological Findings After, (F. H. Verhoeff) xlv, 5; Late Infection, and Enucleation, (Dunbar Roy) xlvii, 42
Iris, Coloboma of, (Jonas S. Friedenwald) liv, 349; Detachment of Anterior Layers of, (Mark J. Schoenberg) lvi, 528; Essential Atrophy of, (G. E. De Schweinitz) lvi, 10; Heterochromia of, (F. Herbert Haessler) liv, 298; Heterochromia of, and Heterochromic Cyclitis, (E. C. Ellett) xlvi, 562; No Cicatrization in the, After Operation or Injury, (M. McBurney) xliii, 12; Mesoblastic Leipomyoma of, (F. H. Verhoeff) lii, 132; Radium in Leucosarcoma of, (Sanford Gifford) xlvii, 241;

Iris (*Continued*)
Ring Sarcoma of, (Abram B. Bruner) liii, 162; Sarcoma of, (G. H. Bell) xlvi, 427
Iritis, Amœbic, in Non-Dysenteric Amœbiasis, (Lloyd Mills) lii, 525; Choroiditis and the Tonsil (J. Dunn) xlvi, 446; Discussion on, xlvi, 572; Nodular, from Hensen's Bacillus, Slit-Lamp Studies, (Luther C. Peter) liii, 258
Iritis and Focal Infections, (W. L. Benedict) l, 560

JACKSON EDWARD, The Capsule in Extraction, xlix, 275; "The Ophthalmic Year Book," xlv, 106, xlvi, 608; Tuberculosis of Retinal Vessels, xlv, 552
JACKSON, EDWARD, and FINNOF, WILLIAM C., Sarcoma of Choroid, xlvii, 72
JACKSON, GEORGE H., Two Early American Works on the Eye, lvii, 265
Jackson, The Work of Hughlings, (James Taylor) xlv, 41
JAMESON, P. CHALMERS, Muscle Recession in Squint, with Scleral Suturing, li, 421; Surgery of Corneal Wounds with Iris Prolapse, lv, 465
JANEWAY, HENRY H., Radium in Eye Diseases, xlix, 156
Jaw-Winking Phenomenon, (A. Lutz) xlviii, 144
JEAN, GEORGE W., "Ophthalmoscopic Diagnosis for General Practitioners and Students," xlv, 105
JESS, A., "Die Sympathische Ophthalmie," xliv, 350
JOHNSON, GEORGE LINDSAY, Accommodation, liii, 426; Capsular Extraction, li, 548; "Comparative Anatomy of Reptilian and Amphibian Eyes," lvii, 553; Couching of Cataracts, liv, 466; Oblique Muscles of the Eye, lvi, 272; Optical Illusions, lvi, 465; Operative Treatment of Glaucoma, xliii, 7

KAZUO HIWATIRI, Histology of the Corneoscleral Margin, Superficial Layer, l, 10
KEARNEY, J. A., Cystotome Manœuvre for Capsular Extractions, xlviii, 62; Squint Operation, when Lenses Correct the Deformity, liii, 274
Keratectomy, (C. Foroni) xlvi, 57
Keratitis, Bullous, (Arnold Knapp) lv, 560; Disciformis, (F. H. Verhoeff) xlviii, 449; Experimental Trypanosome, (C. A. Wood, and G. E. De Schweinitz) xlvi, 431; from Infection by Bacillus Coli, (A. C. Macleish) xliv, 403; Interstitial, lv, 282; Interstitial, from Foreign Body Injury, (Edgar S. Thomson) xlix, 340; Interstitial, Organoluetic Reaction in, (Dohrmann K. Pischel) lvi, 249; Late, after Alcoholic Injections of Gasserian, for Trigeminal Neuralgia, xlix, 621; Neuropathic, Pericorneal Neurotomy in, (F. H. Verhoeff) xlv, 148; Punctata, (A. W. Sterling) liv, 432; Punctata, Conjunctival Flaps in, (Albert Brown) lv, 136
Keratoconus, Bilateral Anatomical, (B. Fleischer) xliii, 626; Three Cases of, (Ervin Török and L. D. Medway) lvii, 19
Keratoplasty, (J. W. Burke) l, 147
KEY, BEN WITT, Antidiphtheritic Serum in Hypopyon Keratitis, xlviii, 581; Protein Injections in Severe Eye Infections, li, 471
KING, BASIL, "Routine Examination of the Eye," liv, 417
KING, CLARENCE, Practical Points in Smith's Technique for Intracapsular Extraction, l, 440; Tubercular Iridocyclitis, with Slit Lamp and Tuberculin, lv, 563
KIRBY, DANIEL B., Congenital Oculo-Facial Paralysis, lii, 452, 598; Cultivation of Lens Epithelium "in Vitro," lvi, 460, 499; Excrescences of Descemet's Membrane, liv, 588; Modified Motais for Blepharoptosis, lvii, 327
KIRKPATRICK, H., "Cataract and its Treatment," li, 300
KNAPP, ARNOLD, Adrenalin in Glaucomatous Eyes, l, 556; Bilateral Circumpapillary Chorioretinitis with Detached Retina in Syphilis, l, 242; Bilateral Glioma of Retina with Glioma in Iris, Autopsy, liii, 470; Bullous Keratitis, lv, 560; Capsule in Cataract Extractions, l, 115; Capsular Extraction, xlvi, 27;

KNAPP, ARNOLD—(*Continued*) Carcinoma of Orbit, xlviii, 120; Cataract in Retinitis Pigmentosa, xlvii, 612; Conjunctivo-Keratoplasty for Staphyloma and Fistula of Cornea, xlv, 349; Embolism of Central Retinal Artery in Chorea, xlvii, 450; End Results in Intracapsular Extractions, lv, 257; Frontal Lobe Tumor of Brain, xlvii, 83; Glaucoma in General Vascularized Nævus of Skin, Case with Angiomatous Changes in the Iris, lvii, 219; Glaucoma in Morgagnian Cataract, lvi, 124; Glaucoma in Myopia, lv, 35; Intradermal Test with Uveal Pigment in Traumatic Iridocyclitis, liv, 252; "Medical Ophthalmology," xlvii, 216; Medical Side of Glaucoma, xlv, 544; Metastatic Thyroid Tumor of Orbit, lii, 68; Oil Cyst of Orbit, lii, 163; One Hundred Capsule Extractions, xliv, 1; Operation for Shrunken Cataracts in Adolescents, lvii, 594; Optic Neuritis after Influenza, Spinal Fluid Changes, xlv, 247; Removal of Orbital Tumor with Preservation of Eye in Plexiform Neuroma of Eyelid, xlv, 475; Second Hundred Successive Extractions in the Capsule, after Preliminary Subluxation with the Capsule Forceps, l, 426; Subperiosteal Abscess in Floor of Orbit, lii, 475; Subretinal Exudate Simulating Sarcoma of Choroid, Anatomic Examination, xlviii, 559; Successful Kroenlein, xliv, 660; Successful Orbital Exostosis Operation, lv, 128; The Autotoxic Factor in Sympathetic Ophthalmia, xlviii, 356; Trephining Results, xliii, 121, 154

KNAPP, ARNOLD, and DWYER, JAMES G., Acute Streptothrix Conjunctivitis, xlvii, 497, 508

KOBI, F. E., "Slit-Lamp Microscopy of the Living Eye," 312

KOELLNER, Neoplasm on Corneal Limbus Cured by Mesothorium, xlvi, 130

Koeppe Nodules, Nature of the, (G. S. Derby) lvii, 561

KOFLER, KARL, and URBANEK, JOSEF, "Vereinfachung und Verbesserung der West-Polakschen Traenensackoperation," lvii, 310

KOLLER, CARL, Mydriatics and Miotics, in Hypertension (Glaucoma), l, 550

KREVET, B., "Der Werth der Bindehautdeckung bei Perforirenden Bulbusverletzungen," xliii, 350

Kroenlein, a Successful, (A. Knapp) xliv, 660

KUEMMELL, R., Atrophying Conjunctivitis, xlvi, 147; Increased Tension Due to Burns and Corrosive Injuries, xliii, 50

Lacrimal Abscess, Duct Conservation in, (A. E. Ewing) liii, 31

Lacrimal Gland, Extirpation, of in Epiphora, (C. R. Holmes) xlviii, 323

Lacrimal Sac, Digital Compression of, in Dacryocystitis, (Juan Santos-Fernandez) xlv, 117; Extirpation of, (Allen Greenwood) l, 234; Intranasal Operation for, (J. M. West) lv, 351; Neoplasms of, (Adolph O. Pfingst) lv, 138; Rapid Dilation with Sea Tangle Probes, (Albert L. Brown) lvi, 397; Sarcoma of the, (T. Harrison Butler) xliii, 16

LAGLEYZE, PROF., Honor to, xlvii, 295

LAGRANGE, FELIX, "Atlas d'Ophtalmoscopie de Guerre," xlviii, 210, 212; "Atlas of Orbital Fractures and Eye Injuries in War," xlviii, 210; "Glaucome et Hypotonie," lii, 517

LAGRANGE, HENRI, "La Tuberculose du Tractus Uveal," liii, 631

LAMB, H. D., Blindness in Missouri and St. Louis, lvi, 469; Keloid of Cornea, l, 535; Myxoma of Orbit with Anatomical Findings, lvii, 425; Tear into Ciliary Body from Contusion, lvi, 332

LAMB, R. S., Fibrolysin, xlvi, 367

LAMBERT, WALTER EYRE, Bilateral Septic Retrobulbar Neuritis, xlvii, 609

LANCASTER, WALTER B., Ames' Spectacle Device for Successful Treatment of Cyclophoria, lvii, 332

LANDMAN, OTTO, Dacryops, liv, 592; Tuberculous Iridocyclitis with

LANDMAN, OTTO—(*Continued*) Fatty Degeneration and Infiltration, liv, 261
LANDOLT, E., Operative Treatment of Squint, xliii, 130
LANE, FRANCIS, Persistent Posterior Perivascular Sheath of Lens, xlviii, 572
LANGDON, H. MAXWELL, and JONES, ISAAC H., Eye and Ear in Barany Tests, xlvii, 348
LANIER, L. H., Vaccine Therapy and Autogenous Vaccines, li, 349
LAWSON, SIR ARNOLD, "War Blindness at St. Dunstan's," lii, 515
Lead Shot, Removal of, from within the Eye, (George H. Cross) lvi, 564
LEDERER, R., Tension in Passive and Active Motion of the Eyes, xliii, 53
LEE, T. P., Apparatus for Near Point and Axis of Astigmatism, lvi, 351
LEMERE, H. B., Angioma of Lids and Brow, xliii, 126
LEMOINE, ALBERT N., and MCDONALD, ALEXANDER, Phacoanaphylactic Endophthalmitis, liii, 101
Lens, Annular Traumatic Opacity of, (P. D. Berrisford) xlvii, 616; Antigen, Cataract Treatment with, (Zenas H. Ellis) lvii, 46; Capsule, Exfoliation of, (Arthur N. Alling) lvi, 1; Epithelium, Cultivation of, "in Vitro," (Daniel B. Kirby) lvi, 450, 499; Lamellar Changes of, (E. Blaauw) liv, 305; Persistent Perivascular Sheath of the, (Francis Lane) xlviii, 572; Protein—Isolation of a Third (Gamma) Crystallin, (Earl L. Burky and Alan C. Woods) lvii, 464; Protein—New Method for Preparation of Beta Crystallin, (Earl L. Burky and Alan C. Woods) lvii, 41
Lenses, Weight and Percentage of Solids in Cataractous, (C. A. Clapp) xlv, 574
Lenticonus, Slit-Lamp Study of, Elias J. Marsh) lvi, 128
Lenticonus Posterior, (Henry H. Tyson) lvii, 38
Leprosy of Eye in Hawaii, (Forest J. Pinkerton) lvi, 42
Leukæmia and the Eye, (M. Cohen) xlvii, 61, (Benjamin Sachs) lvii, 474
LEVINSOHN, G., Genesis of Myopia, liv, 434
LI, T. M., Syphiloma and Syphilitic Iridocyclitis, liii, 531
LICHTWITZ, Gout and Gouty Diseases of the Eye, xliii, 25
Lid, Adenoma-Carcinoma of, and Orbit, (H. W. Wootton) lvi, 275, 294; Gumma of Lower, (J. Bruder) xlvii, 201; Perithelioma of Upper, (N. R. Donnell) liii, 411; Sarcoid of, (G. S. Derby and F. H. Verhoeff) xlvi, 312; Thread-Fixation of, in Operations on, (L. de Liebermann and W. J. Werfelmann) lv, 461
Lid Repair, Epithelial In- and Out-lay, (S. Hanford McKee) xlix, 30
Lids, Conjunctiva and Sclera, Melanosis of, with Warts on Iris, (H. and Jonas S. Friedenwald) liv, 51
Lids, Operative Treatment of Traumatic Coloboma of, (L. Blascovics) xlvii, 404
Lids and Brow, Angioma of, (H. B. Lemere) xliii, 126
Lids and Conjunctiva in Refractive and Muscular Errors, (John Green, Jr.) l, 158
Light- and Color-Sense, Apparatus for Testing the, and for Color Mixing, (C. E. Ferrer and G. Rand) lv, 245
Light Fields, Attachment for Taking, (D. N. Dennis) xliii, 624
LINDNER, "Die Bestimmung des Astigmatismus," lvi, 520
Lindner Squint Operation, (Edmund B. Spaeth) liii, 540
Lipæmia of Retina, (M. Cohen) l, 263, (W. F. Hardy) l, 543
LIPPINCOTT, J. A., Binocular Optical Metamorphopsia, xlvi, 397
LLOYD, RALPH L., Metal Suture in Spastic Entropion, liii, 566; Needling Infection, liii, 431; Phantom Intraocular Tumor, xlviii, 489; "Visual Field Studies," lv, 428; X-Ray Therapy in Glaucoma, lvi, 445
LOHLEIN, W., Visual Fields in Glaucoma and Value for Diagnosis, xlv, 51
LOHMANN, W., "Disturbances of the Visual Function," xliii, 467; Exophthalmometer, xlv, 275
LOURIE, O. R., Endocrine Eye Diseases in Women, liv, 469

LOVE, J. M., Angioma of Choroid, xliii, 607
LOWELL, HOLBROOK, Tendon-Muscle Lengthening, and Forceps, xlvi, 224
LOWENSTEIN, ARNOLD, "Die Tuberkulose des Auges," liv, 210
LUCKIESH, M., Protecting Lenses in Industries, xliii, 382
LUEDDE, Vernal Conjunctivitis, xlix, 43
Lumbar Puncture and Spinal Fluid in Eye Diseases, (James B. Ayer) xlv, 9
LUNT, LAWRENCE K., and RIGGS, AUSTEN FOX, Neurotic Disturbances of Eye Function, lii, 313
LUTZ, A., Congenital Enophthalmos, xlvi, 243; Light Pupillary Reflex, with Unusual Reactions, xlvii, 266, 370; Physiological Anisocoria, xlvii, 587; Traumatic Unilateral Vertical Nystagmus, xlvi, 357

MACCALLAN, "Trachoma and Complications in Egypt," xliii, 224; "Ophthalmic Hospitals in Egypt," l, 300
MACHECK, Ptosis Operation, with Fold in Upper Lid, xliv, 539
MACLEISH, Keratitis from Infection, xliv, 403
MACMILLAN, J. A., Metastatic Carcinoma of Choroid, li, 227
Macular Vision, Evaluation of, (A. C. Snell and Scott Sterling) liv, 443
Maculocerebral Familial Degeneration, (S. J. Beach) lvi, 286
MADDOX, ERNEST, Rod Screen Tests, xlix, 229
Maddox Rod, (P. Dolman) xlviii, 503; Optical Defects, (Percival Dolman) xlix, 194
MAGATH, THOMAS BYRD, Variations in the Abducens, xlviii, 67
MAGHY, CHARLES, One Hundred Glaucoma Cases at Moorfields, xlix, 390
MAGITOT, A. P., Ocular Tension and Aqueous, lii, 1
Malingering, Detection of, (E. Török) lv, 338, 383
MARLOW, F. W., Eye Strain in Relation to Functional Neurosis, lvii, 339; Persistent Accommodation Spasm from Latent Hyperphoria, li, 223; "The Relative Position of Rest of the Eyes and the Prolonged Occlusion Test," liii, 407
MARLOW, SEARLE B., A Case of Hemiangioma of the Choroid, lvii, 484; Isolated Paralysis of Inferior Oblique, lii, 12; Non-Operative Treatment of Glaucoma, lvii, 165
MARPLE, WILBUR BOILEAU, In Memoriam, xlv, 611
MARSH, ELIAS J., Slit Lamp in Posterior Lenticonus, lvi, 128
MARTIN, PAUL, and CUSHING, HARVEY, Glioma of Chiasm and Optic Nerves in Their Intracranial Portion, lii, 209
MATTICE, A. F., Metal in Eye after Magnet Extraction, xliii, 20; Radium Removal of Corneal Tumor, and Radium in Ophthalmology, xliii, 237
MAY, C. H., "Diseases of the Eye," xlvi, 610, li, 613, liv, 211
MAYNARD, F. P., "Manual of Ophthalmic Practice," l, 600
McBURNEY, M., Absence of Cicatrization in Iris after Operation or Injury, xliii, 12
McGUIRE, HUNTER H., Uveitis from Appendical Infection, xlix, 500
McKEE, S. HANFORD, Epithelial In- and Out-lay in Lid Repair, xlix, 30
McLEAN, WILLIAM, Experimental Studies in Pressure and Tonometry, xlviii, 23; Tonometry of Glaucoma, xlix, 301; With Barraquer in Barcelona, lii, 460
Medical Curriculum, Place of Ophthalmology in the, (W. Gordon M. Byers) li, 566
MEDING, C. B., One Hundred and Three Extractions (Eighty-three in the Capsule), xlvi, 331
MEEKER, LOUISE H., and COOK, WARD, Preparation of Gross and Microscopic Eye Specimens, lvii, 185, 206
MEESMANN, "Die Mikroscopie des Lebenden Auges," Slit Lamp Atlas, lvii, 317
Meibomian Cysts, Surgery of, (John Dunn) xlviii, 43
MELLER, J., "Augenaerztliche Eingriffe," l, 597; "Ophthalmic Surgery," lii, 621
MELLER, J., and HIRSCH, OSKAR, "Ueber die Rhinogene Neuritis Retrobulbaris," lvi, 518

Meningitis from Punctured Injury of Eye, (C. A. Veasy) xliv, 10
Mesothorium, (Cuperus) xlvi, 126; Treatment, (Koellner) xlvi, 130
Metamorphopsia, Binocular Optical, (J. A. Lippincott) xlvi, 397
Metastatic Ophthalmia After Pneumonia, (Martin Cohen) xlv, 250
MEYER, S. J., Detachment of the Zonular Lamella, lvii, 121
MILLETTE, J. W., Treatment after Cataract Operations, l, 446
MILLS, L., Intracapsular Extraction, xlvi, 344; Amœbic Iritis in Non-Dysenteric Amœbiasis, lii, 525
Molluscum Conjunctivitis, (Harold and Sanford R. Gifford) l, 227
Monocularity, Relation of Headache to Functional, (Albert C. Snell) li, 5
MOORE, R. FOSTER, "Medical Ophthalmology," li, 613; "The Retinitis of Arteriosclerosis, etc.," xlvii, 644
MORAX, VICTOR, "Glaucome et Glaucomateux," lii, 203; "Le Cancer de l'Appareil Visuel," lv, 614
Morgagnian Cataract, Glaucoma in, (Arnold Knapp) lvi, 124
Motor Anomalies of Eyes, (A. Duane) xlviii, 1
Muscles of Eye, in Asthenopia, (E. H. Hazen) xlix, 70
Muscles of Eye and Adnexa, Gross Structural Anomalies of, (Wm. Campbell Posey) liii, 344
Muscles, Recurrent Paralyses of Ocular, and Ophthalmoplegic Migraine, (A. Duane) liii, 417, 436; Shortening without Sutures (R. P. O'Connor) xliii, 368; Supernumerary Orbital, (C. C. Rush and J. P. Schaeffer) xlvi, 524
Muscular Degeneration, Progressive Familial, (Marcus Feingold) xlv, 533
Mycosis, Metastatic Intraocular, (F. H. Verhoeff) lv, 225
Mydriatics and Miotics in Hypertension (Glaucoma), (Carl Koller) l, 550
Myopia, Can Its Development be Arrested and Its Degenerative Changes be Prevented, (Sidler-Huguenin) xlv, 577; Genesis of, (G. Levinsohn) liv, 434
Myopia in Massachusetts Infirmary, (Margaret Gage) xlvi, 151
Myopic Eyes, Glaucoma in, (Arnold Knapp) lv, 35

NANCE, WILLIS O., and PECK, W. H., "Glaucoma, a Symposium," xlv, 104
Naso-Lacrimal Passages, X-Ray Studies of, (D. M. Campbell, J. M. Carter, and H. P. Dour) li, 462
Naso-Lacrimal Surgery, in Ophthalmological Perspective, (Clifford B. Walker) xlix, 585
NEAME, HUMPHREY, and WILLIAMSON-NOBLE, F. A., "A Handbook of Ophthalmology," lvii, 216
Near Point and Axis, Apparatus for, (T. P. Lee), lvi
Needling, Infection after, (Ralph L. Lloyd) liii, 431
Negroes, Eyesight of, in Africa, (J. N. Roy) xlviii, 72
Nervous Disease, The Eyes in, (Ward A. Holden) xlvii, 221
Neuralgia, Trigeminal, (Charles W. Stevens) xlix, 175
Neuritis, Bilateral Retrobulbar from Sepsis, with Blindness, (W. E. Lambert) xlvii, 609; Retrobulbar, from Toxic Thyroidin, (Myles Standish) xlv, 469; Retrobulbar, in a Baby, (T. Harrison Butler) xlviii, 240
Neurofibromatosis with Buphthalmus, (Alfred Wiener) liv, 481
NEWCOMB, MAJOR C., and WRIGHT, R. E., LT. COL., Analytical Observations of the Vitreous Humor of Normal and Glaucomatous Eyes, lvii, 460
NICOLAS, FELISA, Tuberculosis of Conjunctiva, li, 379
Nocardia (Streptothrix), Infection from, (F. E. Woodruff) liv, 11
NOGUCHI, HIDEYO, and COHEN, MARTIN, Bacteriological Studies of an Epidemic of Koch-Weeks Bacillus, xlv, 155; Experimental Cultivation of the Trachoma Bodies, xliii, 116
Nystagmus, Discussion on, xliii, 281; Unilateral Vertical Traumatic, xlvi, 357

OAST, SAMUEL P., Blue Sclerotics and Brittle Bones in Mother and Child, lvii, 254; Traumatic Dislocation of Eye, liv, 191

Oblique Muscles of Eye, (G. Lindsay Johnson) lvi, 272
O'Connor, D. F., Glioma and Atrophia Bulbi, xlvi, 298
O'Connor, R. P., Shortening of Eye Muscle without Sutures, xliii, 368
Ocular Blood Vessels, Effect of Sympathetic on, (F. H. Adler, E. M. Landos, and C. L. Jackson) liii, 239
Ocular Circulation, Local Control of, (Francis Heed Adler) liii, 1
Ocular Disease, from Deficiency Diseases, (Francis Heed Adler), lvi, 593
Ocular Muscle Paralysis, Pathology of, (Will Walter) xlv, 172
Ocular Tension and the Aqueous, (A. P. Magitot) lii, 1
Ocular Tuberculosis, Pathological Study of Cases of, (Mary Knight Asbury) lvii, 603
Ocular and Cerebral Fluids and Blood, Comparative Chemical Studies of, (Martin Cohen, John A. Killian, and Mildred Kamner) lvii, 59
Oculo-Facial Paralysis, (Daniel B. Kirby) lii, 452
Operations, Illuminating Device for, (J. A. Andrews) xlviii, 65
Ophthalmia in Rats, A Study of Food Deficient in Vitamins, (Arthur M. Yudkin) liii, 416
Ophthalmological Congress at Amsterdam, in 1929, lvii, 218, 444
Ophthalmology, Division of, in University School of Medicine, Chicago, (E. V. L. Brown) lvii, 190; Instruction in, (F. H. Verhoeff) xlix, 1, 112; Post-Graduate Teaching of, (Meyer Wiener), xlix, 9, (A. Duane) xlix, 17
Ophthalmology and the Clinician, (Homer E. Smith) l, 165
Ophthalmoplegia Interna from Lead Poisoning, (L. D. Brose) xliv, 26
Ophthamoplegia Internuclearis, (Edward B. Dunphee) lv, 54
Optic Atrophy, in Sclerosis of Circle of Willis, lvii, 650; Tabetic, Value of Differential Pupilloscopy, li, 507, 581; Treatment of, (E. L. Zimmermann) liv, 545
Optic Atrophy and Chiasmal Lesions, (Clifford B. Walker and Harvey Cushing) xlv, 407
Optic Canal, X-Rays, (Harry A. Goalwin) lv, 1
Optic Disk, Tumor at Edge of, (Arthur M. Yudkin) lv, 364, 435
Optic Nerve Abscesses, (Algernon B. Reese) lvi, 265
Optic Nerve, Atrophy of, Hereditary, and X-Ray Findings, (W. Zentmayer) xlvii, 627; Congenital Division of, (C. A. Snead) xliv, 418; Decompression Operations for Diseases of, (A. Bednarski) xliv, 53; Evulsion of, (E. M. Blake) xlvii, 567; Glioma of, (Herbert Wright Wootton) lvii, 477; Kroenlein for Tumors of, (A. Knapp) xliv, 660; Primary Intraneural Tumors (Gliomas) of, (F. H. Verhoeff) li, 120, 239; Relations of Diseases of the, to Posterior Nasal Sinus, (C. W. Cutler) lii, 331; Removal of Orbital Part of, for Intradural Tumor, Preservation of Eye and Motility, (R. G. Reese) xlix, 515; Tumor of Orbital Portion, (M. Cohen) xlviii, 19
Optic Nerve Sheaths, Hemorrhage into, from Cerebral Artery Aneurysm, (F. H. Doubler and S. B. Marlow) xlvi, 533
Optic Nerve and Accessory Cavities, (J. Van der Hoeve) li, 210
Optic Nerve Meninges, Cyst of, (Martin J. Cohen and Louise H. Meeker) lvi, 211
Optic Neuritis, After Influenza, (Arnold Knapp) xlv, 247; Discussion on, lvii, 199; Retrobulbar, with Disseminated Myelitis, (W. A. Holden) xliii, 231
Optic Neuritis from Accessory Sinusitis, (S. T. Hubbard) xlvii, 206
Optic Papilla, Anomalous, with Good Vision, (M. Goldenburg) xliv, 246
Optical Phenomena, Stage for Demonstration of, lvii, 549
Optico-Ciliary Neurotomy for Enucleation, (A. Pagenstecher) xlvii, 409
Orbit, Abscess of the, Resembling Detachment with Anæmic Infarct of Optic Nerve, (Robert Cartwright Cheney) lii, 252; Carcinoma of, (Arnold Knapp) xlviii, 120; Cavernous Lymphangioma of, (H. S. Gradle) xlix, 520; Chloroma of, (Cyril Barnert)

Orbit—(*Continued*)
liii, 454; Oil Cyst of, (Arnold Knapp) lii, 163; Encapsulated Angioma of, (W. Gordon M. Byers) liii, 280; Exostosis of, Removed, (Arnold Knapp) lv, 128; Four Tumors of, Removed with Preservation of Sight, (W. C. Posey) xlvii, 551; Inflammatory Affections of the, lvii, 537; Lymphoid Infiltration of, (G. Coats) xliv, 221; Metastatic Thyroid Tumor of, (Arnold Knapp) lii, 68; Myxoma of, with Anatomical Findings, (H. D. Lamb) lvii, 430; Neurinoma of, (Martin Cohen) liv, 426; Pseudo Inflammatory Tumors of, liv, 397; Pseudo Tumor of, (William L. Benedict and Mary S. Knight) lii, 582; Radium in Melanosarcoma of, (Edward B. Heckel) xlv, 465; Removal of Exostosis of, (Arnold Knapp) lv, 128; Removal of Psammosarcoma of, with Preservation of the Eyeball, (G. E. De Schweinitz) xliii, 469; Removal of Tumor from Apex of, with Preservation of Eyeball, (Arnold Knapp) xlv, 475; Revolver Wound of, (J. A. Spalding) xlviii, 317; Subperiosteal Abscess in Floor of, (Arnold Knapp) lii, 475; Successful Treatment of Tumor of, with Radium, (G. H. Bell and S. Tousey) xlvii, 514

Orbit and Brows, Tumor of, Treated with Radium, (G. H. Bell and Sinclair Tousey) xlviii, 531

Orbit and Ethmoid, Basal Cell Carcinoma of, Operation, and Radium, (John Green, Jr.) lii, 75

Orbit and Eyeball, Rod Palpation of Contents, and Surface of, (Mark J. Schoenberg) lvi, 438

Orbital Cavity, Tuberculoma of, (Dunbar Roy) lii, 147

Orbital Socket, Restoration of the, (P. N. K. Schwenk and Wm. Campbell Posey) xlvii, 576

Orbito-Palpebral Region, Radium in Cancers of, (George T. Pack) lvii, 246

Orientation, Ocular, Its Importance and Dependence on the Muscular System, (E. H. Hazen) xlix, 434

Out-Patient Work in Ophthalmology, (Conrad Berens, Jr.) li, 519

PACK, GEORGE T., Radiation Therapy of Cancers of the Orbito-Palpebral Region, at the Radium Institute in Paris, lvii, 128

PAGENSTECHER, A., Optico-Ciliary Neurotomy, a Substitute for Enucleation, xlvii, 409

Panophthalmitis, Experimental Production Through Blood Stream, (P. Wegeforth) xlviii, 276

Papillitis, Sudden, Followed by Atrophy, X-Ray of Optic Canals, (Clifford B. Walker) liv, 165

Papilloedema, from Sinusitis, Operative Recovery, (G. H. Bell) xlvii, 344; in Neurology, (G. Horrax) liv, 130; in Oto-Laryngology, (Leon E. White) liv, 142; in Ophthalmic Practice, (James Bordley, Jr.) liv, 158; Surgery of, in Brain Tumors, (Charles A. Elsberg) liii, 307, 384; Symptoms, Diagnosis and Course, (Ward A. Holden) liii, 316, 384

Paralysis, Retraction Movements in Ocular, (Arnold Knapp) lvii, 204

PARKER, WALTER R., Double Luxation of Lenses in Exophthalmic Goiter, li, 1; Permanent Cure of Retinal Detachment after Trephining, xliii, 189, 489; Sellar Decompression Operation for Relief of Blindness, in the Two Eyes, of Varying Degree, xlvi, 528; Trephining in Glaucoma, xlvi, 1

PARSONS, SIR JOHN HERBERT, "Diseases of the Eye," lii, 511, lvi, 518; "Introduction to the Study of Color Vision," xliv, 608

Pelvic Organs in Eye Infection, (W. L. Benedict, W. H. Von Lackum, and A. A. C. Nickel) lvi, 116

Pemphigus, Ocular, (Lloyd B. Witham) li, 389

Penal Institutions, Need of Eye Clinic at, (Conrad Berens) xlvii, 448

Perimetry, Form- and Color-Test Object in, (L. C. Peter) xliv, 416; Quantitative, (C. B. Walker) xlvi, 537

PETER, LUTHER C., Conception of Visual Field Changes in Glaucoma, lvi, 337; Form and Color of Test Object in Perimetry, xliv, 46; Nodular Leprous Iritis, Slit-Lamp Studies, liii, 258; "Principles and Practice of Per-

PETER, LUTHER C.—(*Continued*) imetry," xlv, 609 lii, 619; "The Extra-ocular Muscles," lvii, 322; Visual Fields in Glaucoma, xlix, 309
PETERSEN, WILLIAM F., "Protein Therapy and Non-Specific Resistance," li, 301
PFEIFER, R. A., "Myelogenetische-Anatomische Untersuchungen ueber den Zentralen Abschnitt der Schleitung," liv, 619
PFINGST, ADOLPH G., Neoplasms of Lacrimal Gland, lv, 139
PFINGST, ADOLPH O., and GRAVES, STUART, Melanosarcoma of the Choroid in Brothers, l, 431
Phakœrisis, (Ignacio Barraquer) li, 448, 501
Phantom Eye Tumor, (R. I. Lloyd) xlviii, 489
Philippine Eye Cases, (Antonio S. Fernando) lii, 170
Physiological Accommodation, li, 202
PINCUS, F., Bilateral Acute Serous Tenonitis, xliv, 291; Endogenous Gonorrhœal Corneal Affections, xliii, 136
PINKERTON, FORREST J., Eye Leprosy in Hawaii, lvi, 42
PISCHEL, DOHRMANN K., Organo-Luetic Reaction in Interstitial Keratitis, lvi, 249
Pituitary Body, Hypertrophy of, (Ward A. Holden) xlix, 332
Pituitary Body Disease and Glandular Treatment, (G. E. De Schweinitz, and H. W. How) xlvi, 139
Pituitary Disorders, Non-Surgical Treatment of, (Walter Timme) xlix, 268
Pituitary Lesions, Control of Vision in, by Surgical, X-Ray and Radium Treatment, (Charles H. Frazier) l, 247
Polycythæmia and the Eye, (Martin Cohen) xlvii, 192, 203
POSEY, WM. CAMPBELL, Ætiology and Diagnosis of Glaucoma and Control by Miotics, xlix, 293; A Typical Epithelial Dystrophy of Cornea, xlvi, 113; Gross Structural Anomalies of the Eye and Its Adnexa, liii, 344; "Hygiene of the Eye," xlviii, 207; Melanosarcoma of Conjunctiva, lv, 131; Orbital Tumors Removed with Preservation of Vision, by Incision Along External Orbital Rim, xlvii, 551; Tenotomy of Inferior Oblique, xlv, 137; Unusual Changes in Visual Fields, xliv, 507
POST, M. H., Glaucoma and the Nasal Ganglion, l, 317
Post-Operative Infection, Prevention of, by Diet, (George Huston Bell) lii, 436
Pregnancy, Visual Fields in, (C. A. Finlay) lii, 50
Presbyopia, (E. Fuchs) li, 21, 63
Pressure, Intraocular, (L. Bothman and Seymour J. Cohen) lvi, 110
PRINCE, A. E., Frozen Carbon Dioxide for Trachoma, Vernal Catarrh, and Pterygium, xlv, 219
Projection and Double Vision, (A. Duane) liv, 233
Proptosis, Unilateral, from Probable Ethmoid Osteoma, (Robert Sattler) xlvii, 68
Protecting Lenses, (M. Luckiesh) xliii, 382
Protein Therapy, Specific and Non-Specific, (Alan C. Woods) lvii, 488
Pterygium, (Fumio Yano) l, 239
Pterygium Operation, (L. W. Crigler) li, 577
Ptosis, Living Sutures in Treatment of, (W. W. Wright) li, 99; Operation for, with Fold in Upper Lid, (Macheck) xliv, 539; Shortening of Levator and Tarsus, (L. De Blascovics) lii, 563
Puncta Lacrimalis, Congenital Atresia of One, (Harry Gradle) l, 349
Pupillary Reflexes, Examination of the, (H. E. Gradle) xliii, 377
Pupillary Reflex, Unilateral Argyll-Robertson Pupil with Bilateral Consensual Reaction, (Anton Lutz) xlvii, 266, 509
Pupillary Symptoms in Embolus of Central Retinal Artery, Especially as to Primary Reflex, (John Dunn) xlix, 191
Pupilloscopy, Differential, (Otto Barkan) li, 29
Pupils, Cat-like in Man, (D. Greef) xliii, 146; Closure of, by Cysts and Solid Masses from Uveal Layer of Iris, (J. E. Weeks) xliii, 483; Light Response and Argyll-Robertson Pupil, (John Dunn) xlvi, 193

Purpura Hæmorrhagica, (Martin Cohen) lii, 471

QUACKENBOSS, A., Sclero-Corneal Trephining in Glaucoma, xliii, 600

QUICK, DOUGLAS, Radium and X-Rays in Tumors of Hypophysis, xlix, 256

Quinine Amblyopia, (John E. Weeks) xlv, 342

Rabbits, Syphilitic Infection of Eyes of, (W. H. Brown) li, 408

Radium, in Ophthalmology, (G. Allen Robinson) lv, 328; Uses for, in Ophthalmology, (Henry H. Janeway) xlix, 156; Uses for, xlix, 238, 334

RADOS, ANDREW, Nutrition of Eye, lvi, 567

RAMSAY, A. MAITLAND, "Clinical Ophthalmology," l, 301; Lamp for Ophthalmic Surgery, li, 152

Red Cross Work Amongst the French, l, 302

REESE, ALGERNON B., Abscesses of Optic Nerve, lvi, 265

REESE, ROBERT G., Kroenlein, with Preservation of Eyeball and Motility, xlix, 515; Operation for Blepharoptosis, liii, 26

Reflecting Protective Lenses, (J. Imre) lvi, 141

Reflex Arc, Pupilloscopic Findings in Different Districts, (Francis Heed Adler) lv, 262

Refraction, Changes in Forty Years, (C. F. Clark) lvi, 52; One Hundred Cases of, lvii, 540; Work, (Alexander Duane) xlix, 349

Retina, Air Embolism of Vessels of, (Hans Barkan) lvii, 402; Angiomatosis of, (Hunter W. Scarlett) liv, 183; Bilateral Detachment of, in Two Successive Pregnancies, (Emory Hill) liii, 137; Bilateral Glioma with Glioma in Iris, with Autopsy, (Arnold Knapp) liii, 470; Declination of the Vertical Meridians of the, (G. T. Stevens) xlv, 205, 438, 521; Detachment of, (Derrick T. Vail) xlix, 553; Detachment of, (H. W. Wootton) xlvii, 207; Detachment of, Discussion on, xlix, 639; Double Detachment of, in Nephritis of Pregnancy and Replacement, (E. M. Blake) li, 586; Embolism of Central Artery of, in Chorea, (Arnold Knapp) xlvii, 459; Glioma of, Apparently Cured by Radium with Preservation of Normal Vision, (F. H. Verhoeff) l, 450; Glioma of, Cured in Non-operated Eye by Radium, (M. J. Schoenberg) xlviii, 485; Glioma of, in Non-Enucleated Eye Cured by Radium, (Mark L. Schoenberg) lvi, 221; Glioma of, with Hypotony in Diabetic Coma, (Martin Cohen), l, 247; Gliosis of, in Corneal Staphyloma, (Peter A. Soudakoff) liv, 288; Operation for Detachment of, (Elschnig) xliv, 163; Reduced Silver Nitrate for Sections of, (M. Fernandez Balbuena) lii, 358; Resection of Sclera in Detachment of, from High Myopia, (E. Török) xlix, 506; Scleral Resection in Detachment of, (E. Török) xlvi, 466; Sclerotomy with Drain in Detachment of, (Mayer Wiener) liii, 368; Trephining and Aspiration in Detachment of, (E. S. Thomson) xlix, 563; Vascular Lesions of, in Youth, (H. Friedenberg) liv, 111

Retina and Cerebellum, Case of Hemiangioma of, (Lindau's Disease), (Harvey Cushing and Percival Bailey) lvii, 447

Retinal Artery, Bilateral Vascular Loop of (A. M. Yudkin) lvi, 474

Retinal Hemorrhages in Bothriocephalus Anæmia, (Martin Cohen) liii, 362

Retinal Neoplasms, Natural History of, lvii, 532

Retinal Vessels, Tuberculosis of, (Edward Jackson) xlv, 552

Retinitis Albuminurica, Atypical, (C. A. Clapp) xlix, 98

Retinitis Pigmentosa, Cataract in, Pathology and Treatment, (Arnold Knapp) xlvii, 612

Retinochoroiditis, Photographic History of Traumatic, (Arthur J. Bedell) lvii, 262; Syphilitic (F. H. Verhoeff) xlv, 352

RIEDEL, ALFRED H., Spontaneous Hypotonus in Juvenile Glaucoma, l, 457

RILEY, HENRY ALSOP, Conjugate Deviation with Hemiastereognosis, xlviii, 340, 371

Ring Magnet, (T. Harrison Butler) xlix, 247
RIVERS, W. C., "Consumption and Squint," xlvii, 215
ROBERTS, WILLIAM HUMES, Blindness of Nasal Origin, liii, 19
ROBINSON, G. ALLEN, Radium in Ophthalmology, lv. 328
ROBISON, J. MATHEWS, Blocking of Fifth and Seventh Nerves in Extraction, liii, 570
Rod Screen Test, Letters Concerning, (E. Maddox) xlix, 229
RODIN, Iridocyclitis Treated with Subconjunctival Atropine and Epinephrin, lvi, 277
ROEMER, PAUL, "Lehrbuch der Augenheilkunde," lii, 523
ROENNE, HENNING, Mechanics of Squint Operation, lvi, 428, 503
ROETTH, A., Phacoanaphylactic endophthalmitis, lv, 103
ROLLET, E., and COLRAT, A., "Tuberculose de l'Œil et de ses annexes," lvi, 516
ROMAINS, JULES, "Eyeless Sight," liv, 108
Rosacea Ocularis with Fascicular Keratitis, (Uribe-Troncoso) xlvii, 1
ROWE, E. H., Vaccine Therapy in Choroiditis from Alveolar Abscess, xlvi, 251
ROY, DUNBAR, Epithelioma of Eyeball and Lids, xlviii, 563; Late Infection needing Enucleation after Iridotasis in Glaucoma, xlvii, 42; Tuberculoma of the Orbital Cavity, lii, 147
ROY, J. N., Eye Diseases amongst African Negroes from Syphilis and Trypanosimiasis, l, 28; Eyesight of Negroes in Africa, xlviii, 72
RUSH, C. C., and SCHAEFFER, J. P., Supernumerary Orbital Muscles, xlvi, 524

SACHS, BENJAMIN, The Eye in Leukæmia, lvii, 474
Salvarsan and Other Remedies in Ophthalmology, (Freeland Fergus) xlvii, 28
SANTOS, FERNANDEZ, Digital Compression of Sac, in Dacryocystitis, xlv, 117
Sarcoma, Epibulbar, with Sections, (L. W. Crigler) xliv, 41; Positive Focal Tuberculin Reaction in a Perforating, (J. W. Charles) xlviii, 568
SARTAIN, PAUL J., On Lewis Ziegler, lv, 423
SATTLER, H., "Boesartigen Geschwuelste des Auges," lv, 615
SATTLER, ROBERT, Acute Tularense Conjunctivitis, xliv, 265; Penetrating Injury of Eyeball with Subsequent Tetanus, xlvii, 64; Unilateral Proptosis Operation, Probable Ethmoidal Osteoma, xlvii, 68
SAVAGE, G. C., "Nervo-Muscular Mechanism of the Eye, and Routine in Eye Work," xlvi, 611; "Ophthalmic Neuro-Myology," lv, 429
SCARLETT, HUNTER W., Angiomatosis of Retina, liv, 183; Diplobacilli in Corneal Ulcers, xlvi, 219
SCHAEFER, J. PARSONS, "The Nose and Olfactory Organs," xlix, 549
SCHIFF-WERTHEIMER, SUSANNE, "Les Syndromes Hemianopsiques," etc., lvi, 313
SCHIRMER, O., Optic Hallucinations with Hemianopsia, xlvi, 247
SCHOENBERG, MARK J., Acute Retrobulbar Neuritis in a Child with Intestinal Stasis, xlviii, 56; Bilateral Glioma of Retina Cured by Radium, lvi, 221; Detachment of Anterior Layers of Iris, lvi, 538; Diagnosis and Treatment of Luetic Involvement of Optic Pathways, xlix, 204; Ephedrin in Eye Practice, lvii, 272; Glioma Apparently Arrested with Radium, xlviii, 485, 522; Remarks on Mydriatics and Miotics in Hypertension, li, 156; Rod Palpatory Examination of Orbital Contents and Surface of Eye, lvi, 438; The Knapp Adrenalin Mydriasis Reaction in Descendants of Glaucoma Patients, liv, 282
SCHOENBORN, Basedow, Tetany and Acromegaly, xliv, 421
SCHWENK, P. N. K., and POSEY, WILLIAM CAMPBELL, Restoration of Orbital Socket, xlvii, 576
Scleritis, Brawny, (George S. Derby) xlv, 20; Cause and Pathology of Brawny, (W. Gilbert) xliv, 545
Sclerostomy, Histology after, (F. H. Verhoeff) xliv, 129

Sclerotics, Blue and Brittle Bones in Mother and Child, (Samuel P. Oast) lvii, 254, 304
Sclerotomy, with Relation to Late Infection, (T. H. Butler) xliv, 611
Scotoma, Tangent Plane for Measuring, (A. Duane) xliii, 420
Scotomata, Scintillating, (J. A. Spalding) xlviii, 38
SHANNON, J. R., Aneurysm of Internal Carotid, and Effects on Vision, xlvi, 518
SHARPE, W., Fundus Pressure Signs from Intracranial Conditions, xlvi, 320
SHINOBU ISHIHARA, "Tests for Color Blindness," xlvii, 436
SIDLER-HUGUENIN, Can the Development of Myopia be Arrested, and Its Degenerative Changes Prevented?, xlv, 577
SIEGRIST, A., "Refraction und Akkommodation des Menschlichen Auges," liv, 618
SINSKY, H. L., LEVIN, M. B., and SACKS, B., Episcleritis, Diet Treatment, l, 526
Sinusitis, Ocular Involvement in, (Edgar S. Thomson) lvii, 653; Paranasal, Eye Manifestations of, (James Bordley, Jr.) l, 137
SISSON, R. J., Basophilic Staining of Bruckner's Membrane, lv, 125
Skull, Eye Manifestations in Fracture of, (M. Cohen) xlvi, 258
SLATAPER, S. J., Normal Reserve Accommodation in Presbyopia, lv, 370
Slit-Lamp Attachments, lvii, 307
Slit-Lamp Studies, lv, 72, 276
Slit-Lamp Research, (Basil Graves) lv, 319
Slit Lamp, Uses of, (H. S. Gradle) li, 264
Slit Lamp and Blood Vessels, liii, 410
SLUDER, GREENFIELD, "Concerning Some Headaches and Eye Disorders of Nasal Origin," xlviii, 208; "Nasal Neurology, Headache and Eye Disorders," lvii, 316
SMITH, DORLAND, Cavernous Sinus Thrombosis, xlvii, 482; Choice of Cataract Operations, lii, 25
SMITH, HENRY, Acidosis Œdema as Related to Glaucoma, xlv, 1; Congenital Cataracts, Drawn up Pupil after Extraction, Lid Control in Extractions, Cataract Treatment, l, 422, 468, 514; Early Senile Cataract, Ptosis and After-Cataract, li, 40; Extraction in the Capsule, lv, 213; The Iris in Cataract Extraction, lvi, 105
SMITH, HOMER E., "Applied Refraction," lvi, 422; Ophthalmology and the Clinician, l, 165
SMITH, JAMES W., Melanosarcoma of Choroid, lvi, 156
SNEAD, C. N., Congenital Division of Optic Nerve, xliv, 418
SNELL, ALBERT C., Compensation for Eye Injuries, liii, 37, 172; Headache and Functional Monocularity, li, 5; Injuries of Superior Oblique, xlviii, 111
SNELL, ALBERT C., and STERLING, SCOTT, Percentage Evaluation of Macular Vision, liv, 443
Snellen-Symbol, and Visual Value, lv, 447
SNYDER, WALTER H., Observation, Management and Treatment of the Cataract Patient, Before and After the Operation, l, 120
SOUDAKOFF, PETER S., Gliosis Retinæ in Staphylomatous Corneæ, liv, 288
SPAETH, EDMUND B., Lindner Squint Operation, liii, 540; "Newer Methods of Ophthalmic Plastic Surgery," liv, 617
SPALDING, J. A., Revolver Bullet Injury of Orbits, xlviii, 317; Scintillating Scotomata, xlviii, 38
SPICER, W. T. HOLMES, "Parenchymatous Keratitis, Interstitial Keratitis, Uveitis Anterior," liii, 295
Sporotrichosis, Ocular, (S. R. Gifford) li, 540, liii, 264
Squint, Muscle Recession and Suturing, (P. C. Jameson) li, 421; Operations for, (E. Landolt) xliii, 130; Treatment of, (Arthur Whitmire) lii, 242
Squint Operation, Mechanics of, (Roenne), lvi, 429, 503
Squint Operation when Lenses Correct Deformity, (J. A. Kearney) liii, 274
STANDISH, MYLES, Retrobulbar Neuritis from Toxic Thyroidin, xlv, 469
STARK, H. H., Effects of Syphilis on Eye Injuries, xliv, 49; Vitreous Opacities, xlvi, 38

Stereopsis, Routine Measurement of, (David W. Wells) xlix, 64
Stereoscopic Vision, Device for Examining Form and Colors (M. Cohen) xlviii, 142
STERLING, SCOTT, and SNELL, A. C., Percentage Evaluation of Macular Vision, liv, 443
STEVENS, CHARLES W., Facial Herpes and Faulty Eye Muscle Adjustment, liii, 60, 74; Trigeminal Neuralgia, xlix, 175
STEVENS, GEORGE T., Declination of the Vertical Meridians of the Retina, xlv, 205, 438, 521
STIRLING, A. W., Abiotrophy, Ophthalmoplegia Externa, lii, 56; Influence of Age on the Axis of Astigmatism, l, 19; Keratitis Punctata, liv, 432; Law of Rotation of Astigmatic Axis, lii, 62
STIRLING, J. W., Blockage of Trephine Opening, xlvi, 352
STOKES, JOHN H., Syphilotherapy and the Eye, lvi, 229
Streptococci, Localization of, in the Eye, (E. V. L. Brown) xlv, 229
Sub-Retinal Exudate, Simulating Sarcoma of Choroid, (Arnold Knapp) xlviii, 559
SUKER, G. F., Tarsus Excision in Trachoma, xlvi, 210
Superior Oblique, Injury of, (A. C. Snell) xlviii, 111
Supra-Sellar, Cyst, Removed, (H. S. Gradle) lv, 431
SWANZY, "Handbook of Diseases of the Eye," xlix, 549
SWEET, WILLIAM M., Implantation of Metal Ball, xlviii, 257
Sympathetic Ophthalmia, Anaphylaxis in, (A. C. Woods) xlvi, 8; Autotoxic Factor in, (Arnold Knapp) xlviii, 356; Blood Count in Diagnosis of, (S. D. Gifford) xlvii, 327; Cases Cured, (Derrick T. Vail), li, 255; Diphtheria Antitoxin in Treatment of, (Edward B. Heckel) lvii, 54; Successfully Treated with Vaccines, (Alfred Wiener and Ellis Bonime), l, 43, 97; Uveal Pigment in, (Alan C. Woods and Arnold Knapp) li, 560
Synchysis Scintillans in Anterior Chamber, (John N. Evans) liii, 142
Syphilis, Effect of, on Injuries of the Eye, (H. H. Stark) xliv, 49
Syphilis and Iritis, (H. Dickson Bruns) liv, 462
Syphilitic Eye Diseases, Literature of, (Stanley O. Chambers) lvii, 412
Syphilitic Involvement of Optic Pathways, (Mark J. Schoenberg) xlix, 204
Syphilitic Lesions of Eye, (J. C. Hopkins) lvi, 500, 543
Syphilitic and Trypanosomiatic Eye Diseases Amongst African Negroes, (J. N. Roy) l, 28
Syphiloma and Syphilitic Iridocyclitis, (T. M. Li) liii, 531
Syphilotherapy, (John H. Stokes) lvi, 229
SZILY, A., "Anaphylaxis in Ophthalmology," xliv, 353

Tangent Curtain, (A. Duane) xliii, 591
Tarsus, Turning of, (L. De Blascovics) liv, 38
TASSMAN, I. S., Proteins of Lens and their Chemical Changes in Pathogenesis of Senile Cataract, lvii, 362
TAYLOR, JAMES, The Work of Hughlings Jackson, xlv, 41
TAYLOR, ROBERT HENRY, "Glaucoma," xlviii, 203
Teeth and Eye, xlviii, 85
Tendon Muscle Lengthening and Forceps, (H. Lowell) xlvi, 224
Tendon-Tucker and Silk Suture, (Frank E. Burch) li, 360, liv, 333
TENNER, A. S., Tabetic Optic Atrophy, and Value of Differential Pupilloscope, li, 581
Tenonitis, Serous, (G. N. Brazeau) lv, 152; Serous Bilateral, (E. Pincus) xliv, 291; Serous with Bilateral Papilledema, (G. N. Brazeau) lii, 355
Tenon's Capsule, Embryology of, (Isadore Goldstein) lii, 327; Implantations into, (W. M. Sweet) xlviii, 257
Tension, Increased, from Injuries and Burns, (R. Kuemmel) xliii, 50; in Active and Passive Motion of the Eye, (R. Lederer) xliii, 53; in Motions of the Eyeball, (R. Lederer) xliii, 53; Narrowing Pupil does not Lower Normal Intraocular, (Andy M. Carr) xlvii, 177

TERRIEN, F., "Chirurgie de l'Œil et de ses Annexes," l, 595
Test Cards for Visual Acuity, (Alfred Cowan) lvii, 283
Tetanus Following Limited Penetrating Injury of Eyeball, (R. Sattler) xlvii, 64
Third Nerve Paralysis, Correction of Defect due to, (Meyer Wiener) lvii, 597
THOMA, KURT H., "Oral Roentgenology," xlvii, 436
THOMASSON, A. H., Chronic Gonorrhœal Prostatitis a Possible Cause of Ocular Inflammations, lii, 546; Development of Glaucoma Scotoma, lvi, 319; Tangent Screen in Field Taking, lv, 545; Value of Scotometry in Diagnosis and Treatment of Glaucoma, lvii, 160
THOMSON, EDGAR S., Trephining and Aspiration in Retinal Detachment, xlix, 563
THORINGTON, JAMES, "Methods of Refraction," xlv, 610
TIMME, WALTER, Dispituitarism and Glandular Treatment, xlvi, 151; Non-Surgical Treatment of Pituitary Tumors, xlix, 268
Tonometer with Mercury, (M. Cohen) l, 326
Tonometric Chart, (T. Harrison Butler) xlix, 227
Tonsil Disease and the Sight, (H. F. Hansel) xlvii, 572
Tonsillectomy, Tension in Normal Eyes Before and After, (Andy M. Carr) xlvii, 46
TOOKE, FREDERICK, Corneal Trephining for Glaucoma, li, 14
TOOKER, C. W., and LAMB, H. D., Iridocyclitis Sympathica, Following Iridectomy for Secondary Glaucoma, liii, 439
TÖRÖK, E., Malingering, lv, 338; Resection of Sclera in Detachment from High Myopia, xlix, 506; New Iridectomy Technique in Glaucoma, lii, 574; Scleral Resection in Detachment, xlvi, 466; Tuberculin and Eye Diseases, xlviii, 242
TÖRÖK, ERVIN, and REDWAY, L. D., Cases of Keratoconus, lvii, 19
Torticollis and Tenotomy of Inferior Oblique, (Alexander Duane) xlv, 33
TOUSEY, SINCLAIR, and BELL, GEORGE HUSTON, Radium Treatment of Brow Tumor, xlviii, 531
Toxæmias, Effect of, on the Eyes (W. H. Wilmer) xlix, 461
Toxins and Protein Degeneration Products, Action of, on the Eye, (Alan C. Woods and James L. Stoddard) xlv, 321, 451
Trachoma, in Palestine, (Arieh Feigenbaum) lv, 159, 453; Nature of, and Normal Histology of Conjunctiva, (Kazuo Hiwatari) xlix, 82; Silver Nitrate for, (N. R. Donnell) liii, 433; Tarsus Excision in, (G. F. Suker) xlvi, 210; Vernal Catarrh and Pterygium, Frozen Carbon Dioxide for, (A. E. Prince) xlv, 219
Trachoma Bodies, Cultivation of, (Noguchi and Cohen) xliii, 117
Trachoma Studies, (Noguchi) lvi, 423, 503
Trephining, Histology of Eyes Lost from Complications Following, (W. G. M. Byers) xlvii, 462; Priority Correspondence, xliii, 225, 353; Return of Tension and Retro-Choroidal Hemorrhages after, (Arnold Knapp) xlvii, 256, 308; Sclero-Corneal, One Hundred Cases of, (A. Quackenboss) xliii, 600; Some Results after, (A. Knapp) xliii, 121, 154
Trephining Orifice, Blockage of, (J. W. Stirling) xlvi, 352
Tuberculin Treatment, (E. Török) xlviii, 242, 295
Tuberculin in Ocular Tuberculosis, (P. Berghausen) lvii, 583
Tuberculosis, Anterior Ocular, (G. S. Derby, and Maud Carvill) lvi, 523; Clinical Aspects of Ocular, (W. H. Wilmer) lvii, 1; Syphilis and Neoplasms of Eye from Industrial Trauma, (Hans Barkan) li, 102; Ocular, Experimental and Clinical, (William C. Finnoff) liii, 130
Tumor, Melanotic Epibulbar, Cured by Radium, (William H. Wilder) liii, 355
Tumors, Diagnosis of Intraocular, (E. E. Blaauw) lv, 159
Typhoid Inoculations, with Eye Complications, (F. Phinizy Calhoun) xlviii, 553
TYSON, HENRY H., Lenticonus Posterior, lvii, 38
TYSON, H. H., and SCHOENBERG, M. J., Blood and Aqueous Changes after Wood Alcohol, xliv, 275

UHLENHUTH, EDUARD, Influence of Function on Structure of Eye, xlvii, 401
Ultra-Violet Light, Protection from Harmful Effects of, (W. E. Burge and A. J. Neill) xliv, 498
URIBE-TRONOSCO, Eye Changes in Acne Rosacea, Fascicular Keratitis, xlvii, 1
Uveal Tract, Bilateral Sarcoma of, lvii, 543; Value of Uveal Pigment in Diagnosis of Injuries of the, (Alan C. Woods) li, 451, Value of, in Treatment of, (Alan C. Woods and Arnold Knapp) li, 460
Uveitis, Pathology of, (S. R. Gifford) li, 483; from Appendical Infection, (Hunter H. McGuire) xlix, 500; Sympathetica, Diphtheritic Antitoxin in, (F. H. Verhoeff) lvi, 28; Thyroid Treatment of Malignant, (J. Bordley) xliv, 638

VAIL, DERRICK T., Conjunctivitis Tularense, lv, 235; Detachment of Retina, xlix, 553; Modified Elliot-Lagrange Technique in Glaucoma, lii, 346; Sympathetic Ophthalmia Cured, li, 255; Uncomplicated Cataracts, xlv, 307
VAIL, DERRICK T., JR., and VERHOEFF, F. H., Endophthalmitis, Retained Foreign Body Phagocyst, lvi, 5
VALOIS, G., "Les Borgnes (One Eyed) de la Guerre," xlviii, 213
VANDEGRIFT, GEORGE W., Optics of Cornea, xlix, 421
VAN DER HOEVE, J., Eye, Ear, and Vestibular Organs, li, 321; Optic Nerve and Accessory Cavities, li, 219
VEASEY, CLARENCE A., Neuritis and Glaucoma after Herpes Zoster, xlviii, 364; Purulent Meningitis from Fish-hook Injury of Eye, xliv, 10
VERHOEFF, F. H., Capsule Forceps for Intracapsular Extractions, xlv, 479; Cyclectomy in Glaucoma, liii, 228; Diphtheritic Antitoxin in Sympathetic Uveitis, lvi, 28; Eye Operations, lvi, 385; Glioma of Retina Treated with X-Rays with Apparent Destruction of the Tumor and Preservation of Normal Vision, l, 450; Histological Findings after Iridotasis, xlv, 5; Histological Findings after Sclerostomy, xliv, 129; Keratitis Profunda, xlviii, 449; Mesoblastic Leiomyoma of Iris, lii, 132; Metastatic Intraocular Mycosis, lv, 225; Microscopic Findings in Coralliform Cataract, with Remarks on Congenital Cataracts, xlvii, 558; Mycoisis of Choroid after Extraction, and Metastatic Choroiditis Resembling Sympathetic Uveitis, liii, 517; Pathogenesis of Glaucoma, liv, 20; Phacoanaphylactic Endophthalmitis, lv, 296; Primary Intraneural Tumors (Gliomas) of Optic Nerve, li, 120, 239; Suture for Cataract Extraction, lvi, 137; Syphilitic Retinochoroiditis, xlv, 352; Teaching of Ophthalmology, xlix, 1; Various Forms of Neuropathic Keratitis, Pericorneal Neurotomy, in, xlv, 148
VERHOEFF, F. H., and DERBY, G. S., Plasmoma of Lacrimal Sac, xliv, 252
VERHOEFF, F. H., and FRIEDENWALD, JONAS, Blepharochalasis, li, 554
VERHOEFF, F. H., and SISSON, R. J., Basophilic Staining of Bruch's Membrane, lv, 125
VERHOEFF, F. H., and WAITE, J. H., Separation of Choroid, lv, 38
Vernal Conjunctivitis, (W. H. Luedde) xlix, 43
Vestibular Nerve Problem, Phases of, (J. Dunn) xlvii, 354
Vision, Improved by Decompression, (W. Parker) xlvi, 528; in Light of Reduced Intensity, (Carsten Edmünd and H. Ulrik Moeller) liv, 531; Its Economic Value and Conservation, (L. B. Witham) xliv, 523; Percentage, and Snellen Notation, (J. A. C. Gabriels), lv, 58; the Rôle of Pigment in the Physiology of, (Francis Heed Adler) lvii, 346
Visor, Cruise's Protecting, xlvi, 478, xlvii, 594
Visual Acuity, A New Unit of, (L. De Blascovics) liii, 476
Visual Acuity and Illumination, (Selig Hecht) lvii, 564
Visual Complex, (Abram B. Bruner) liv, 372
Visual Field in Optic Neuritis, (J. M. Griscom and Margaret M. Monroe) lvi, 244

Visual Fields, Bi-Temporal contraction of, in Pregnancy, (C. A. Finlay) lii, 50; Changes in, from Vascular Lesions, (W. C. Posey) xliv, 597
Vitiligo, Ocular Depigmentation with Generalized, (David Wheeler) lvii, 393
Vitreous, Hemorrhages, Ciliary Epithelium in Absorption of, (J. Meller) lvii, 134; Magnetic Removal of Foreign Bodies from, (Lee Masten Francis) xlix, 198; Opacities, (H. H. Stark), xlvi, 38; Prevention of Loss of, in Intracapsular Extractions, (W. A. Fisher) xliv, 560; Value of Aspiration of, (Zur Nedden) lvii, 109
Vitreous Humor, Observation of, from Normal and Glaucomatous Eyes, (Major C. Newcomb and Lt. Col. R. E. Wright) lvii, 480
VOGT, ALFRED, and VON DER HEYDT, "Atlas of Slit Lamp, Microscopy of Living Eye," li, 197

Wafers, Minus Tri-Focal, (Talbot R. Chambers) li, 154, 172
WAGENER, HENRY P., and GIPNER, JOHN D., Visual Fields in Localized Chorioretinitis, lv, 470
WAITE, J. HERBERT, Glaucoma from Intraocular Fluid Exchange, lvi, 460, 522
WALKER, CLIFFORD B., Abrupt Moncular Blindness, Double Papillitis, Optic Atrophy During Encephalitis, liv, 165; Bitemporal Hemianopsia, Instruments for, xliv, 369; Naso-Lacrimal Surgery, xlix, 585; Quantitative Perimetry, xlvi, 537; Wilbrand Test Prism Phenomenon with an Instrument, xliv, 109
WALKER, CLIFFORD B., and CUSHING, HARVEY, Optic Nerve Atrophy and Chiasmal Lesions, xlv, 407; Visual Field Distortions in Brain Tumor and Chiasmal Lesions as Related to Hemianopsia with Hypophyseal Tumor, xlvii, 119
WALTER, WILL, Clinical Pathology of Ocular-Muscle Paralysis, xlv, 172
WANDLESS, H. W., Subconiunctival Flap Extraction with Capsulotomy, xliii, 494
War, Eye Phenomena in Psychoneuroses of, (G. E. De Schweinitz) xlviii, 419; Eye Work in France, (G. S. Derby) xlviii, 217
Wassermann Test, Symposium on, li, 493
WEEKS, J. E., Glaucoma after Herpes Zoster, xlvi, 460; Occlusion of Pupils by Cysts and Masses from Uveal Layer of Iris, xliii, 483; Operative Treatment of Glaucoma, xlix, 316; Quinine Amblyopia, xlv, 342
WEGEFORTH, PAUL, Experimental Panophthalmitis from Bloodstream Infection, xlviii, 276
WELLS, DAVID W., Measurement of Stereopsis, xlix, 64; "The Stereoscope in Ophthalmology," xlvii, 437
WEST, J. M., Intranasal Lacrimal Sac Operation, lv, 352
WEXLER, DAVID, Ocular Degeneration with Generalized Vitiligo, lvii, 393
WHEELER, JOHN M., Plastic Operations for War Injuries of Eyelids, xlix, 35; "Surgery of the Eye," xlv, 103
WHITE, JAMES W., Spasm of Accommodation, xlvii, 466
WHITE, LEON E., Papilloedema in Oto-Laryngology, liv, 142
WHITMIRE, ARTHUR, Correction of Squint, lii, 242
WHITNALL, S. ERNEST, "The Anatomy of the Human Orbit and Accessory Organs of Vision," li, 300
WIENER, A., Dacryocystorhinostomy, xlvi, 227; Device for Lateral Homonymous Hemianopsia, lv, 362; Epiphora, xlviii, 334; Neurofibromatosis with Buphthalmus, liv, 481
WIENER, ALFRED, and BONIME, ELLIS, Sympathetic Ophthalmia Successfully Treated with Vaccine, l, 43, 97
WIENER, MEYER, Correction of Defect from Third Nerve Paralysis, lvii, 597; Postgraduate Teaching of Ophthalmology, xlix, 9; Posterior Sclerotomy with Permanent Drain for Detachment, liii, 368
Wilbrand Test with a New Instrument, (C. B. Walker) xliv, 109

WILDER, WILLIAM H., Melanotic Epibulbar Tumor Cured by Radium, liii, 355
WILLIAMS, ANNA, and ROSENBERG, CAROLYN, Purulent Conjunctivitis in Infants, Causes and Effects on the Vision, xlv, 109
WILMER, W. H., Effects of Toxæmias on the Eye, xlix, 461; Ocular Condition Affecting the Aviator, xlvii, 439; Ocular Functions of Aviators, xlviii, 439; Ocular Tuberculosis, lvii, 1; Sclerocorneal Trephining in Glaucoma, xlv, 333
WITHAM, LLOYD B., Ocular Pemphigus, li, 389
WOELLFLIN, E., "Tafeln zum Nachweis von Rot- und Gruensichtigkeit," lvi, 312
WOLFF, JULIUS, Conjunctival Apron in Cataract Extractions, lii, 554
WOLFF, JULIUS, and GLOBUS, JOSEPH, Foreign Protein for Retrobulbar Amaurosis, lvi, 576
Wood Alcohol, Changes in Blood and Aqueous after Inhalation of, (H. H. Tyson and M. J. Schoenberg) xliv, 275
WOODRUFF, F. E., Nocardia (Streptothrix), liv, 11
WOODS, ALAN C., Action of Toxins and Protein Degeneration Products on the Eye, xlv, 321; Immunology in Ophthalmology, liii, 312; Ocular Anaphylaxis, xlvi, 8, 283, 503; Ocular Anaphylaxis, Reaction of Perfusion, with Specific Antigen, xlv, 557; Ocular Anaphylaxis, Experimental Iridocyclitis, xlvii, 161; Protein Therapy, Specific and Non-Specific, lvii, 488; Uveal Pigment in Diagnosis of Injuries of the Uveal Tract, li, 451
WOODS, ALAN C., and DE SCHWEINITZ, G. E., Trypanosome Experimental Keratitis, xlvi, 431
WOODS, ALAN C., and KNAPP, ARNOLD, Therapeutic Use of Uveal Pigment in Sympathetic Ophthalmia, li, 460
WOODS, ALAN C., and STODDARD, JAMES L., Action of Proteins and Protein Degeneration Products on the Eye, xlv, 321, 451
WOODS, CASEY A., "Fundus Oculi of Birds," xlvi, 610
WOODS, HIRAM, Lethargic Encephalitis, xlviii, 536
WOOTTON, HERBERT WRIGHT, Adenoma Sarcoma of Upper Lid and Orbit, lvi, 275, 294; Glioma of Optic Nerve, lvii, 377; Non-Surgical Treatment of Heterophoria, iv, 440
WORTH, CLAUD, "Squint, Its Causes, Pathology, and Treatment," xlv, 103
WRIGHT, R. E., Amyloid Degeneration of Conjunctiva, liv, 280; Blocking of Facial Nerve in Extractions, lv, 555; Conservative Treatment of Staphyloma of Cornea, liv, 55; Facial Nerve Blocking in Cataract Extractions, lii, 166; Parinaud's Syndrome (?), liv, 423; "Report of Government Ophthalmic Hospital at Madras for 1922," lii, 620, liii, 624
WRIGHT, W. W., Living Sutures in Ptosis, li, 99

Xanthomatosis, (Sanford R. Gifford) lvi, 281
X-Rays, Ophthalmic, (G. S. Dixon) lvii, 653

YUDKIN, ARTHUR M., Congenital Tumor at Edge of Optic Disk, lv, 364, 435; Rat Experimental Ophthalmia with Vitamin-Free Rations, liii, 416; Vascular Loop of Retinal Artery, lvi, 474
YUDKIN, A. M., KRAUSE, A. C., and MORTON, D. G., Transmission of Arsenic to Anterior Chamber, lvii, 17

ZENTMAYER, W., Etiology of Hereditary Optic Nerve Atrophy, X-Ray Findings, xlvii, 627
ZIEGLER, S. LEWIS, Memoir of, lv, 423
ZIMMERMANN, E. L., Arsphenamines in the Production of Ocular Lesions, lvii, 509; Iridocyclitis and Its Influencing Factors, liii, 558; Tabetic Optic Atrophy, liv, 545
Zonular Lamella, Detachment of, (S. J. Meyer) lvii, 121
ZUR NEDDEN, Aspiration of Vitreous, lvii, 109

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